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## DUODENAL ULCER: APPRAISAL OF CURRENT METHODS OF MEDICAL TREATMENT*


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One of the striking present day medical phenomena is the prevalence of duodenal ulcer. Many competent observers at home and abroad are of the opinion that this disease is definitely on the increase throughout the civilized world. The nature of treatment of such a disorder, affecting as it does the health, efficiency, and life of many adult members of every community,[10] should be of universal interest. It is now estimated that 5 per cent or more of people have peptic ulcer at some time during their lives, and that more than 375,000 individuals consult physicians each month throughout the United States because of disorders arising from ulcer. Yet, only about 1 out of every 4 ulcer-bearing patients has disturbances of sufficient degree to consult physicians or to be hospitalized.

Both physicians and surgeons are generally agreed that uncomplicating gastric ulcer and duodenal ulcer, particularly the latter, should be treated medically. The trend from surgical to medical management is shown in figure 1. Lahey and Marshall, in 1943, reported operating on 6.5 per cent of 6.500 patients who had duodenal ulcer and 18 per cent of 450 patients who had gastric ulcer. The trend and percentage of patients with duodenal ulcer who underwent operation at the Mayo Clinic from 1928 through 1942 is shown in figure

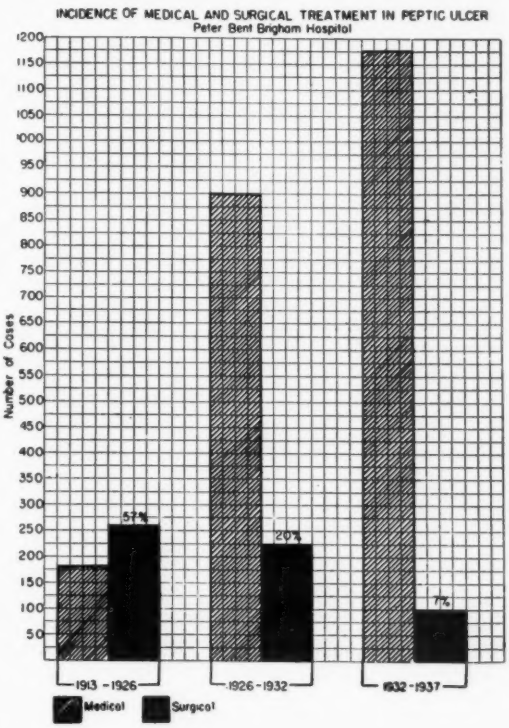


Fig. 1. Trend from surgical to medical management of peptic ulcer in a representative Eastern hospital. (From Zollinger, Robert: The Surgical Aspects of Peptic Ulcer. Rhode Island M. J. 21:113 (Aug.) 1938.

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2. Up to and including 1946, 60 per cent of the patients with gastric ulcer were

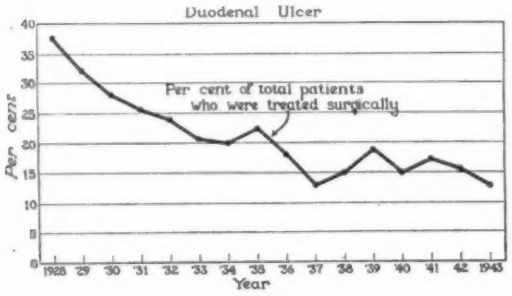


Fig. 2. The trend, from 1928 through 1943, of the percentage of patients with the diagnosis of duodenal ulcer who were treated surgically at the Mayo Clinic.

submitted to operation. However, in the past two years there has been a sharp decrease, only about 35 per cent being operated on in 1947. The tendency toward increasing conservatism in the case of uncomplicated ulcers is evident in spite of the uncompromising attitude of some surgeons, particularly with respect to gastric ulcers.

As on many other points in the realm of gastroenterology, authoritative opinion is divided as to whether the patient who has duodenal ulcer shall be treated in ambulant fashion or be sent to the hospital. There are those who maintain that hospitalization; is not essential in the majority of cases, while others regard it as highly desirable and rather insist on it as a routine procedure. Treatment and observations in the hospital are indicated, as a rule, under the following circumstances: when there is a coexisting gastric ulcer; when some complication, such as gross bleeding, exists or is impending; when there is gastric retention or threat of perforation of a penetrating lesion; when the pain is severe or more or less persistent; when the patient is highly nervous, exhausted, or hypersensitive, or it is felt that he will not adhere strictly to an ambulatory regimen; when it is essential that he or she be relieved of the cares of office or home, for only in this way can physical and mental rest be secured and effective treatment be carried out at the same time. However, the fact remains that the majority of patients are treated in ambulant fashion, and with the rising cost of hospital care, the shortage of beds and nurses in the United States during and since World War II, more and more patients necessarily will be treated in ambulant fashion. Although the average cooperative patient who has a relatively mild uncomplicated ulcer does very well when treated in ambulant fashion, in my judgment, the previous treatment of the majority of patients who come under my observation has been inadequate. One is reminded of Moynihan's criticism that the failure of medical treatment was attributable to its insufficiency and that few patients received any treatment which offered a reasonable prospect of healing the ulcer. This is regrettable because proper and adequate treatment instituted at an early stage of the disease is the best guarantee of permanent cure with the least expenditure of time, effort and money.

The first objective is to heal the lesion and the second objective is to keep it healed or prevent a recurrence at the same site or elsewhere. Not only is the latter more difficult to obtain, as a rule, but its importance has not been sufficiently stressed in the past. Whether ulcer is regarded as a separate entity, or as the local manifestation of a general systemic derangement, the patient, as a whole, must be encompassed in the therapeutic planning. This, apart from a complete physical inventory, presupposes tactful inquiry into the psychosomatic and environmental aspects, and into matters of personal habits and daily mode of eating, working, and sleeping. In a given case, the degree and baneful effect of emotional tension, physical fatigue, focal infection, alcohol, tobacco, strong tea and coffee, condiments, stimulating or coarse foods, improper mastication from whatever cause, dysfunction of the bowel or associated disease of other viscera must be determined. Continued cooperation with respect to diet, medication, personal and mental hygiene is usually achieved only by fully acquainting the patient with the na-

ture of his disease and with the rationale underlying treatment. There is much justification for the old adage: "Once an ulcer patient, always an ulcer patient." The tendency for the patient to throw off all restraint after he has experienced comfort for several weeks or longer must be zealously guarded against; otherwise, recurrence is in the immediate offing.

### CHOICE OF METHOD OF TREATMENT

Because of disagreement concerning its cause and the fact that prevailing methods may not be completely satisfactory, innumerable cures have been suggested for ulcer. Many of these so-called cures are in vogue for a short time and then are discarded as their predecessors have been. Like purported cures for cancer and tuberculosis, past experiences have repeatedly taught me to be just as suspicious of ulcer cures as of the former. I also look with disfavor on parenteral methods of treatment of the ordinary uncomplicated duodenal ulcer. The evaluation of unorthodox forms of treatment is difficult at times because some ulcers heal spontaneously, and many heal after little treatment. Moreover, the clinical course of the disease is characterized by variable periods of remission and exacerbation, and such remissions, especially if prolonged for one reason or another, could easily lead to a false appraisal of the value of the type of treatment instituted. Symptomatic relief is no criterion of healing. There must be anatomic restoration as well. The bulbar deformity of a duodenal ulcer of several years' duration may persist after complete healing has taken place, although the crater (niche) of such an ulcer when present should have disappeared.

Recent progress in the treatment of ulcer, in Winkelstein's opinion, has been characterized by the introduction of aluminum preparations, continuous intragastric drip, protein hydrolysates, the psychosomatic approach, and vagotomy. As vagotomy is purely a surgical procedure, it will be omitted from present considerations. The logical therapeutic procedure is one that takes into consideration the results of experimental research in the production of ulcer, the morbid physiologic processes in the presence of an active duodenal ulcer and the lessons gleaned from clinical observation on the part of physicians and surgeons alike. Hence, the majority of physicians subscribe to the principle of a bland, soft, and nutritious diet, frequent feedings, antacids, temporary use of sedatives and antispasmodics, physical and mental rest and other auxiliary measures whenever indicated. Although the role of corrosion or digestion by the acid gastric juice is unlikely the sole one in pathogenesis, the effectiveness of neutralizing agents should not be minimized. Dragstedt has recently re-emphasized the noteworthy fact that the medical management of duodenal ulcer has been successful directly in proportion to the degree in which the acid gastric juice has been neutralized during the entire twenty-four hours. Successful surgical treatment is based on the same premise.

### CHOICE AND EFFECT OF ANTACIDS

The use of nonabsorbable preparations of aluminum hydroxide in the form of a liquid gel or tablet has replaced the former use of the salts of sodium, calcium, magnesium, and bismuth capable of systemic absorption and their hazards. Aluminum hydroxide also has adsorptive, bactericidal, mild astringent, and demulcent properties. The degree of neutralization of gastric acidity which is necessary for ordinary purposes is achieved at the so-called proteolytic neutralization point; that is, at a pH of 4.5 to 5.0. Complete neutralization requires an elevation of the pH to at least 7. Adequate neutralization throughout the day and night is exceedingly difficult to attain in 80 per cent of cases except by continuous intragastric drip. In spite of this fact and without continuous intragastric drip the majority of patients experience relief and the ulcer frequently heals. Voegtlin has recently called attention to the difficulty of reducing the nocturnal acidity by the usual methods, but has stated that healing may proceed nevertheless.

My choice of an aluminum hydroxide preparation for the past five years has

been the gel or tablet in combination with magnesium trisilicate. The investigations of Batterman and Ehrenfeld have demonstrated that the addition of the latter increases the effectiveness of the former and decreases its constipating effect as well. This preparation in tablet form is for the convenience of the patient when away from home or in the office. Another tablet as a convenient substitute for the inter-meal milk feeding is one made of compressed skim milk in combination with calcium carbonate.* The continuous drip treatment using aluminum hydroxide gel or alkalinized milk is employed only for patients who have refractory ulcers and are tolerant to such method.

### INDICATIONS FOR, AND CHOICE OF, SEDATIVES AND ANTISPASMODICS

Since many ulcer-bearing individuals are a nervous, tense, hyperreactive, and irritable lot, their sleep is not restful and usually they are mentally and physically exhausted. While treatment in the hospital affords complete rest and relaxation which is conducive to healing, nevertheless, the circumspect use of sedatives and antispasmodics is indicated from the outset to facilitate recovery. For obvious reasons, such medication is even more imperative for that great majority of patients treated in an ambulatory way. While rest, dietary measures, antacids, and frequent feedings per se relieve the pain and characteristic gastric neuromuscular disturbances in whole or large part, sedative and spasmolytic agents should hasten and insure such action.

Belladonna and its alkaloids, atropine, hyoscyamine, and scopelamine, have been prescribed for centuries by the medical profession as antispasmodics in the treatment of gastrointestinal disorders. In more recent years, a variety of synthetic preparations bearing familiar proprietary labels have been manufactured. Several more recent synthetic preparations have a combined analgesic and antispasmodic effect. A variable combination of sedatives and antispasmodics also is available in tablet form, and aluminum hydroxide preparations in gel or tablet form have been made in combination with synthetic antispasmodics and sedatives. Thus the modern physician has a much wider range of such preparations to choose from now than formerly.

Certain precautions are necessary in the use of all drugs of this nature. The idiosyncrasy of many persons toward belladonna and its derivatives is well known. Moreover, the amount of sedation required to achieve the desired effect without impairing the patient's efficiency is a variable one. As Kilstein has observed, the problem is that of proper dosage of phenobarbital or its equivalent with suitable antispasmodics in harmonious combination. This dosage and combination not only should provide a maximum of effect but should be in such proportions that it can be used for a prolonged period if that should prove necessary. One of my favored combinations is a powder or copsule containing 1/4 grain (16 mg.) of phenobarbital; 1/24 grain (2.7 mg.) of homatropine methyl bromide and 20 grains (1.3 gm.) of calcium gluconate.

### TREATMENT WITH PROTEIN HYDROLYSATES (AMINO ACIDS)

Levy and Siler, in 1942, suggested, and Co Tui and his associates developed, the use of amino acids. While the proponents of this form of treatment have advocated it for all ulcers, I have never seen the justification except when hypoproteinemia, as a result of hemorrhage, pyloric obstruction, or malnutrition from whatever cause is present, assuming the patient can tolerate a preparation which decidedly has an unpleasant taste which is difficult to disguise. Protein deficiency in the patient with an uncomplicated ulcer is usually absent or minimal. Additional proteins are of secondary importance in treatment if the patient's diet otherwise is adequate. After judicious and impartial appraisal of other aspects than those already mentioned, namely, buffering value, effect on gastric motor function, immediate and late clinical results, it seems safe to predict that protein hydro-

*Nutrachloric tablet (Upjohn).

lysate therapy per se will not enjoy a widespread and sustained popularity.

### DIETETIC TREATMENT

The keystone in the therapeutic arch is a proper diet. At the clinic we subscribe to the principle of (1) food devoid of chemical, mechanical, and thermal irritation, (2) food of bland nature readily evacuated by the stomach, and (3) the taking of such food at frequent intervals, particularly in the earlier stages of treatment in order to minimize or control the corrosive action of the gastric juice, especially in the interdigestive phase. Vitamin concentrates and preparations of iron also are prescribed whenever the indication exists. At stated intervals, depending on the progress made, an increase in the amount and variety of the food is permitted. At the clinic we have devised a booklet[5] which is helpful in instructing patients.

### TREATMENT FOR AMBULANT PATIENTS

The reader should consult the textbooks by Bockus, and by Eusterman and Balfour, or the chapter by Palmer in Portis' textbook, for a description of the details of treatment in the hospital. At the clinic the ambulant patient undergoing treatment is instructed to start either on the "B" or "C" diet, as outlined in the booklet just mentioned. The diet chosen depends on the severity and duration of the symptoms. The "B" diet consists of three small meals of cereals, cream soup, simple desserts, and orange juice, and 120 cc. of milk and cream each hour in addition. At intervals of one to two weeks the diet is increased successively to the "D" diet and then to the regular ulcer regimen for ambulatory patients. For the first two or three months the patient takes 30 cc. of a mixture of aluminum hydroxide and magnesium trisilicate in gel or tablet form one hour after each meal and at 11 a. m., 4 p. m., and bedtime. At intervals of two hours from 7 a. m. to 9 p. m., 120 to 180 cc. of whole milk is consumed for a similar period. The regular "ambulatory ulcer diet" is followed for a year. After the patient has been using this diet for three or four months the dose of aluminum hydroxide and magnesium trisilicate is decreased to 15 cc. or one tablet. A sedative-antispasmodic preparation of the physician's choice is taken from the outset fifteen minutes before the three regular meals and at bedtime for a period of two to four weeks. The degree of success achieved is often directly in proportion to the cooperation given by the patient.

### SUMMARY

Considerations pertaining to treatment of duodenal ulcer should command universal interest because of the ubiquity and increasing incidence of this disease throughout the civilized world. The trend has been from surgical to medical management of the uncomplicated duodenal ulcer in the past two decades. While the majority of patients are ambulatory during treatment, the indications for and advantages of treatment in a hospital are also discussed.

In the past, treatment in most cases has been inadequate. Proper and adequate treatment at an early stage of the disease is the best guarantee of permanent cure. To heal the lesion is the first objective and to prevent a recurrence is the second. This second aspect is more important, is difficult to attain, and is often neglected.

The introduction of preparations of aluminum hydroxide, continuous intragastric drip, the psychosomatic approach, and probably vagotomy and protein hydrolysates, has characterized recent progress in the treatment of ulcer. Arguments for the choice of the method of treatment as prescribed by the writer are advanced. A brief outline of the procedure for the ambulant form of treatment is included.

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# SURGICAL VIEWPOINT OF LEG ULCER WITH PARTICULAR REFERERENCE TO POST-THROMBOPHLEBETIC ULCER

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NEW ORLEANS

Leg ulcers have been more or less despised by the surgeon and have been regarded by the profession in general as 'dressing problems. The disadvantages of this attitude to the patient have been numerous and discouraging. The hope of a patient for a cure has been permitted to languish and disappear. Ultimately such patients learned to tolerate an open infected sore, often painful and malodorous. Many new advances have been made, however, and in reality the prophylactic and active treatment of leg ulcer today is justifiably and increasingly more optimistic. More can be done to prevent these severe complications,—for complications they are, —and more can be done to actually cure them.

In order to comprehend the problem in general it is necessary to view it systematically, divided into various components. I have made a classification of indurations of the leg and leg ulcers, based on etiology.[1]

Read at the meeting of the Orleans Parish Medical Society, July 12, 1948.

The advantages of such a classification may be obvious. The factors responsible for an ulcer should be considered and then attention should be directed to the alleviation of of the individual cases. Since in the majority of instances a number of different factors are operating to produce the ulcer, several different therapeutic measures may be indicated. If one simply removes one of the causes, only a 25 per cent factor in the ulceration, a cure should not be expected. It is easy to direct attention to one cause of an ulcer and to overlook others which may be even more potent in producing the vitiated physiology responsible for that lesion.

The classification which I have outlined includes under nine different headings all of the various causes which may operate either individually or collectively to produce leg ulcers. They may be enumerated as follows:

1. Post-thrombophlebitic edema.
2. Varicose veins.
3. Obesity.
4. Trauma.
5. Infection, either pyogenic or fungous.
6. Lymphedema.
7. Arteriovenous communications.
8. Immersion leg.
9. Systemic causes.

When viewed as a problem of removing one or a number of these various factors in a patient with a leg ulcer the entire question becomes very complex, involving an extent of knowledge and experience and technical ability which will tax the surgeon

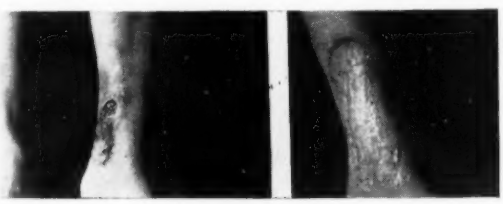

Fig. 1. Post-thrombophlebetic edema ulcer in a young woman, aged 27 years. The deep vein thrombosis occurred in a puerperium six years ago. The ulcer has been present for two years. It was very painful. A lumbar sympathetic ganglionectomy was performed followed by excision of the entire indurated area and skin graft. The patient has remained well now for one year.

in order to obtain satisfactory results for the patient. This makes it all the more interesting, and when the various different therapeutic measures which may be used systematically or locally, and the operative procedures which may be indicated, are all considered, the modern surgical aspects of leg ulcer become an intricate engaging problem. Today leg ulcer is certainly something more than a dressing problem.

I have seen leg ulcers, for which the patient came as a primary complaint, attributable to a toxic nodular goiter with cardiac decompensation. Trauma, also, in

Fig. 2. The results of bilateral excision of indurated areas for bilateral post-thrombophlebetic edema ulcers. The patient aged 58 had had recurrent ulcers on one leg for thirty years and on the other leg for ten years. She had suffered great pain with the ulcers which previously had been treated as dressing problems. She has remained well for over one year.

these instances may be an additional factor. I have seen leg ulcers attributable to a disturbed venous return circulation which in turn was caused by funnel chest. The proper solution of the problem in these instances then becomes one of thyroidectomy and operative correction of the deformed sternum. Various other systemic causes, whether medical or surgical, can thus be the real problem for solution of leg ulcer.

One of the most common leg ulcers presenting itself in private work is post-thrombophlebitic edema ulcer. My experience, now over a period of some twenty years of intense interest with vascular disease, has led me to certain crystallized conclusions regarding this problem, conclusions which frequently do not coincide with the opinions usually expressed in the literature. At other times they are in definite agreement with such expressed opinions. One of the most controversial of all the problems today is that of deep vein thrombosis of the lower extremity. From a clinical standpoint two different aspects must be considered: (1) the danger of pulmonary embolism which exists following a thrombosis of the veins of the lower extremity; (2) the residual, a persisting edema in the lower extremity which may ultimately lead to ulceration. The problem of pulmonary embolism has received frequent consideration in the literature in recent years. To prevent pulmonary embolism ligation and section of the femoral vein or the iliac vein has been advocated rather forcefully by certain authors. The profession in general apparently has taken a relatively conservative attitude towards this. Moreover, it now becomes apparent that anticoagulants alone can produce results statistically superior in the salvage of life to the results following operative transsection of the femoral vein.[2, 3] Perhaps heparin and dicumarol will soon replace most of the surgery done to prevent pulmonary embolism. It is my personal opinion that ligation and transsection of the femoral vein never can be done without permanently damaging the return circulation in the lower extremity. I know this is not acknowledged by most authors. On the other hand, in those instances in which I have transsected the femoral vein, and in those cases that I have encountered subsequent to its performance elsewhere there has always been a diminished return circulation which created either a manifest or latent edema of the lower extremity. Many of these patients will not show a manifest edema simply because they are not called upon under stress to use their legs. If they must work eight to twelve hours a day the leg on which the ligation has been done will most assuredly show edema whereas the normal leg will not show it. Statistics themselves are misleading. They have been used to

point out the immediate salvage of life following operative transsection for deep vein thrombosis in the lower extremity. Like many statistics they are not overwhelmingly convincing. Moreover these statistics never seem to take into account the subsequent life of that patient, after he has been saved. Many of these individuals become chronically partially disabled because of swelling and pain in the lower extremity and sometimes because of ulceration. They may be unable to work long hours or if they are young they may be unable to meet successfully the severe competition they find on every side during life. For this reason, some consideration should be given as to whether ligation of the femoral vein is an advantage to the patient in his subsequent life, not just a question of a slightly improved chance to save his life at the time of a fresh thrombosis. Is it better to salvage 1 or 2 more people out of 200 by imposing on 50 out of 100 a certain amount of increased invalidism? I take a very conservative attitude towards ligation of the femoral vein. I believe that it is indicated at times and my criteria as to the indications for surgical interruption of the femoral vein to prevent pulmonary embolism are: first, after a patient has had a minor embolus, to prevent the occurrence of a grave embolus which may subsequently follow; second, it may be employed to diminish the expected post-thrombophlebitic edema if the patient has had a very extensive thrombosis with massive edema. Transsection of the vein and removal of this embolus may permit better development of collaterals and thus insure a subsequent life with less post-thrombophlebitic edema than would be present if the massive long thrombosis was permitted to remain in the vein itself. Shorter less extensive thrombi may more adequately be taken care of by anticoagulants. This is a clinical viewpoint which I have carefully and critically observed and believe to be true. There is no attempt to prove it statistically.

Usually the immediate factor which leads up to leg ulcer is edema. The prevention of this edema is highly important. The skin in and around the internal malleolus of the leg has less vitality, less metabolic activity than any skin of the body. When one considers how rapidly wounds of the face and neck heal and how slowly those heal on the lower extremity in the region of the ankle, this fact becomes apparent. Wounds heal slowly in the region of the ankle, and other pathologic processes such as abscesses retrogress slowly. No matter what the cause of the edema, it should be prevented from accruing day after day. If trauma has occurred it is going to take longer for the results of the trauma, that is the edema and swelling or laceration, to heal and dissipate than it would if the same amount of reaction to trauma had occurred on the upper extremity. When the patient is up the recurrence of daily edema is facilitated, and it is necessary to have them interrupt the consecutive hours of dependency of the lower extremities. It is not altogether the total hours a patient is up during the day but the number of consecutive hours which fosters the accumulation of edema in the lower extremity. For that reason interrupted rest and elevation of the leg, when a cause for edema exists, will prevent subsequent hardening and induration of the subcutaneous tissues, which ultimately may lead to an ulcer. When one cause of edema is present, such as a deep vein thrombosis, then the patient, following the establishment of a new cause, such as infection or trauma, must be doubly cautious and must repair to bed for fear that the combination of the two causes will increase the thickening of the subcutaneous tissue and cause pigmentation and dermatitis and ultimately ulceration. It is much easier to prevent ulceration than to cure it.

What then, is the method of controlling edema following thrombosis involving deep veins of the lower extremity? It is my view that these patients should immediately be told that they will be six months to a year recovering from this complication. Then I insist on measures which "keep them under the edema level." If they are conscientious, if they do not permit the leg to swell day after day, if they are persistent

in taking interval rests with their leg elevated during the day at first, never missing a midmorning and a midafternoon period, the results are usually excellent. Adequate return circulation establishes itself after a long time. After a year they may expect to resume completely normal activity with no evidence of daily recurring edema. On the other hand, if the patients, accepting an idea that ultimately the swelling will diminish spontaneously, stay up continuously day after day and permit recurrent swelling, the intercellular spaces become large and are ready to receive edematous fluid again the next day; thickening and scarring of the subcutaneous tissue results and the leg never can return to normal.

Immediate therapeutic measures which already have been mentioned should not be overlooked. They will not be discussed in detail. Foremost among these are anticoagulants, particularly heparin in a Pitkin's menstruum, ligation in certain instances, with removal of the clot, and various other measures, including sympathetic nerve blocks with procaine. They may be helpful and proper in certain cases. No one measure should be used to the exclusion of all others. These may take the patient over the immediate phase, but the long continued clinical, or subclinical edema which results day after day is another phase which doctors have relegated to the patient. The doctors should recall this responsibility, instruct the patient how to avoid it, and thus ultimately obtain a good collateral circulation which will be adequate to carry on an active life.

When post-thrombophlebitic edema ulcer has occurred then a variety of measures have been suggested for its relief. Various types of dressings and local applications have been used, such as gentian violet, zinc peroxide, Unna paste boot, compression bandages, sulfonamides, penicillin ointment, dried red blood cells, tyrothricin, chloresium and numerous other measures. Some of these are helpful. Let me here endorse dried red blood cells which seem to have a definite effect both in cleaning the granulations and in stimulating epetheliazation. But no amount of local application will heal the ulcer when the factors which are responsible for it are permitted to be maintained.

Sympathectomy has been advocated for this condition. I have performed it in a number of cases. I believe there is a place for it. In younger individuals with a mild dermatitis and weeping of the skin and daily recurring edema, sympathectomy seems to help tremendously at times. It dries the skin, improves the arterial circulation and seems to diminish the venous stasis. I have been disappointed with it in older individuals, and it certainly is not a dramatic cure in itself for post-thrombophlebitis edema or for post-thrombophlebitic edema ulcer. It may be a helpful aid in certain cases. Another measure which has recently been advanced by Buxton and Coller[4] is transsection and ligation of the femoral vein in long standing cases of old deep vein thrombosis. I mention this to condemn it. It is my opinion that transsection of these veins, just as one would expect, makes the condition worse. Moreover, at the same time, I would like to condemn ligation of varicose veins when the patient has had a definite history of deep vein thrombosis. I have gone through the disappointment following such a ligation a number of times. I have felt that even though the patient has had a deep vein thrombosis the varicosities with their incompetent veins and retrograde flow must be additional factors in the edema and ulceration, and thus have been tempted to conclude that ligation without the use of sclerosing solution may be helpful. Everytime I have tried it I have regretted it. I would advise strongly against the ligation of the saphenous vein when it is varicose, when there has been a previous thrombosis of the femoral vein with daily recurring edema. Those of you who insist on trying this yourself, if open minded and critical of your own results, I believe, will ultimately come to the same opinion which I have established. My experience leads me to believe that one should not transsect more veins after a deep vein thrombosis.

In reality you would expect the physiology not to be improved and such clinically, not from laboratory experimentation but from a clinical observation, is actually the result. The theory that transsection and ligation of the deep vein after it has been thrombosed will obviate the spasm in other vessels and thus permit better collateral return, or that it lessens hydrostatic pressure in the deep system, does not prove tenable from clinical experience.

One last measure which I wish to endorse is a very modern dramatic advance in the surgical management of post-thrombophlebitic edema ulcer. For these instances where induration has occurred, and it practically always occurs above the internal malleolus on the inner and lower third of the leg, the tissue which is involved is the subcutaneous tissue and skin. The circulation seems to be adequate for all other parts of the extremity, including the foot. It is surprising but edema following deep vein thrombosis usually does not affect the foot. This may be attributed to the fact that the return circulation from the foot goes up the deep veins and from the inner side of the leg it returns through the long saphenous system. The measure which we have found most acceptable, most dramatic and most helpful is to excise the tissue which does not have adequate venous return, that is the skin and subcutaneous tissue and the fascia in the area above the internal malleolus. This includes the ulcer and the hard indurated area. Then a split graft is placed down onto the muscles and onto tissue which has a more normal return circulation. This graft, then, without edematous fat, and fibrous subcutaneous tissue is able to withstand the demands put on it by daily dependency. It has adequate return circulation and will remain viable. We have obtained excellent results by this method in at least 90 per cent of the cases. These patients' legs are healed and remain healed. The idea of excising an ulcer and putting a graft on it is old; the concept that the removal of all tissue which does not have adequate return circulation is the optimum method of managing post-thrombophlebitic induration and ulceration may be new. I believe that this concept will gain importance in the surgical management of this condition. Sometimes in these cases, in order to secure a dry leg and avoid a weepy dermatitis frequently associated with these cases, we perform a sympathectomy as an adjunct measure.

Numerous other factors responsible for these ulcers should be considered. Neither space nor time permits me to go into detail concerning our viewpoint on varicose ulcer, traumatic ulcer, and ulcer due to obesity and infection. Practically the same attitude regarding these is at present our concept regarding the therapeutic problem. The entire problem becomes very complex. There are many surgical measures to be used. A lot can be accomplished for these patients to restore them to a normal and active existence. For this reason, leg ulcer is no longer just a dressing problem. It is a problem in which brilliant surgical results may be obtained by the properly directed measures.

SUMMARY

A classification of leg ulcer based on causes is given. A new concept of leg ulcers, forming a complex surgical viewpoint, is discussed. Much can be done to alleviate these conditions and to cure these patients permanently. Leg ulcer has been removed from the category of a dressing problem into the category of a surgical problem.

Ligation of either the deep or the superficial venous system of the lower extremity following deep vein thrombosis is condemned. It does harm. A new concept of excision of tissue which cannot be maintained normal by the return circulation, so deficient following a deep vein thrombosis, has been expressed. Excision of ulcers of the lower extremity and skin grafting, removing all tissue not adequately maintained by the circulation, gives brilliant results in over 90 per cent of the cases.

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# GOUT, AN ANCIENT AND NEGLECTED DISEASE*

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"Screw up the vise as tightly as possible, and you have rheumatism," said Dr. Morris Longstreth almost a century ago; "give it another turn and that is gout." The following typical case is illustrative of this disease.

A white male, aged 52 years, had attacks of pain in the first metatarsophalangeal joint of the left foot two or three times a year for ten years. The original attack, however, was first experienced in the left wrist, which became swollen, red, and very painful to touch. At that time his family physician thought that it was due to an insect bite. The attacks were limited to these two joints except for the last attack which involved the right wrist and the right ankle. The patient could usually predict when an attack was impending by a feeling of marked lassitude about twenty-four to forty-eight hours in advance. He responded well to colchicine and aspirin therapy.

The physical examination revealed nothing of consequence. The arterial pressures were 120 mms. of mercury systolic and 80 diastolic, with a cardiac rate of 70 beats per minute. Following the last attack of gout, which was the most severe, he became aware of a small hard area about the size of a number 6 shot located over the right mastoid process. This mass was excised and sodium urate crystals were demonstrated microscopically. Kidney function as determined by the test employing posterior pituitary extract was within normal variation.

Repeated roentgenograms of the left foot failed to reveal any abnormalities for a period of ten years, until his last attack when punched-out areas almost 2 to 3 millimeters in diameter were observed on the head of the metatarsal bone and the base on the proximal phalanx on the left great toe. It is worth emphasizing that a questionable area of calcification in the thoracic aorta was present. Uric acid levels varied from 4.5 mg. per 100 cc. between attacks to 7.5 mg. per 100 cc. during attacks.

Like other more rare diseases in medicine the frequency with which gout is diagnosed depends upon the criteria which are employed. It is, however, generally accepted that from 5 to 8 per cent of all cases of joint disease are due to gout. One may define this disease as a disturbance in metabolism associated with an increase of urates in the body, particularly in and about the joints, with the later development of a chronic deforming arthritis. Deranged purine metabolism plays an important role in this disease. In purine metabolism, the protein is broken down to nucleoproteins and further broken down to nucleic acids by trypsin. It then proceeds through the various secondary but nevertheless highly important stages to nucleotides and to adenine and guanine, and finally to xanthine and uric acid. Because individuals who have gout have uric acid blood levels usually in excess of 6 mg. per 100 cc. of blood, an explanation for gout should account for this increase in blood uric acid. Three possibilities suggest themselves: (1) a diminished destruction of urates by the body, (2) a decreased excretion by the kidney, or (3) increased formation by the body. It is generally accepted that there is no good evidence that urates are destroyed by the body. A decrease in the ability of the kidney to clear the blood stream of urates is accepted by many as the prime cause of the disease, although some authorities, particularly Talbott,[1] think that gout results from an increased production of urates by the body. Both schools of thought, however, agree that individuals who have had gout over any great length of time have impaired renal function. No attempt will be made here to review in detail the pathologic alterations of gout other than to point out that the urate deposits, both subcutaneous and osseous, are the characteristic lesions of gout and that they act as foreign bodies so that the response is one of inflammation. Frequently the joint changes are similar to those of hypertrophic arthritis, on the one hand, and again, may resemble in some features the rheumatoid variety. The kidneys show punctate areas throughout the cortex and in the medulla. Although there may be various forms of

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glomerulonephritis present it is generally accepted that these are coincidental findings. It is worth noting that individuals who have gout exhibit a greater frequency of arteriosclerotic changes both in the general and coronary circulation, and cardiac changes are commonly observed of the type usually found in individuals who have hypertension of long standing.

The relative frequency of the disease has already been discussed. Approximately 95 per cent of the individuals suffering from gout are males, which suggests that the disease is of a sex-linked character although recent work would indicate that about 10 per cent of cases are in females.[4] Since the original description of Sydenham, the age incidence has usually been given as the fifth decade of life, but many cases have been reported in extremes of age and it is not unknown that at 6 to 8 years children may suffer with the disease as well as individuals over 70 years. Although there are many physicians of considerable repute who believe that overindulgence in rich food and alcoholic beverages is an important etiologic agent in the production of gout, evidence is accumulating that there is no preponderance of gout in any social stratum or in relationship to the varieties of food or drink taken. In this respect, gout may be compared with diabetes. Excessive intake of sugar does not cause diabetes, nor does excessive intake of purines or alcohol cause gout, but once the disease is present, excessive intake of the respective foods accentuates the symptoms and signs.

Typically the premonitory symptoms of acute gout are malaise, depression, and mild nonspecific gastrointestinal disurbances which usually precede the attack by twenty-four to forty-eight hours. These are followed by pain in a joint which may occur at any time of the day but frequently is observed at night. In the majority of cases, the metatarsophalangeal joint of the great toe is involved, hence the synonym podagra. After the great toe, and in decreasing frequency, come the other joints of the foot, the knee, the hand, the shoulder, and the vertebrae. Usually one joint is involved at a time although polyarticular attacks do occur, or as one joint subsides another may become involved. The joint swells rapidly, becomes hot, tense, and shiny. The color is that of a deep bluish red. The involved joint is extremely sensitive and the patient frequently describes the pain as though the joint were in a vise. Usually there is an elevation in temperature ranging from as high as 102° to 103° F., with accompanying polymorphonuclear leucocytosis. Towards morning the severity of the symptoms subsides and although the joint may remain swollen, the day is spent in comparative comfort, only to have the symptoms recur the following night. If untreated, the attack usually runs its course in from five to eight days. Diuresis may precede the attack by a day or two. With it, however, there is gain in weight from a decreased loss of insensible perspiration.[5] A drop in the barometric pressure frequently precedes the weight gain and diuresis and these cyclical changes may account for the fact that many patients with gout can predict a day or so ahead of time that they are in for another siege. The prognosis in most patients with gout is a guarded one. The widely used dictum "once gouty, always gouty" is usually true, but it is quite evident that in essence the short term prognosis rests upon the degree of irreversible changes which have taken place particularly in the kidneys and cardiovascular system.

In spite of the very evident inflammation there is never suppuration of the lesion. Usually if the attack has subsided, the patient feels better than he did prior to the attack, and the joint is restored to its normal function. Ordinarily, an individual will experience three or four attacks a year, and with increasing frequency of the attacks, the joint symptoms persist longer and gradually many joints become affected so that finally considerable joint changes take place, and after a number of years, the joint may become swollen, irregular, and deformed. It is, of course, at this stage, that the roentgenogram shows

punched-out defects in the bone but these appear too infrequently to be of great help in the diagnosis of this disease.

Patients with chronic gout almost invariably present evidence of generalized arteriosclerosis with an increase in arterial pressures and cardiac hypertrophy. Kidney function is usually decreased. The volume of urine is increased with a low specific gravity, a few hyalin casts and a trace of albumin. Examination of the cartilages of the ear particularly reveal tophi which, when examined microscopically, or chemically, disclose uric acid crystals.

A complication of gout that is especially deserving of mention is renal stones. Brochner-Mortensen[2] reported that 15 per cent of his patients with gout passed renal stones. Hench[3] states that gouty nephritis affects about 10 per cent or more of gouty patients. Because of this, in a patient with renal colic, gout should be considered as an etiological possibility, and if associated with the renal colic, there is a history of acute or chronic arthritis, the possibility of gout should be thoroughly investigated. It should be remembered that urate stones are not radio-opaque.

It has been the experience of some that an attack of gouty arthritis may be easily provoked by a surgical procedure and other trauma. Linton and Talbott[6] noted postoperative arthritis in 86 per cent of their patients with gout following twenty-two surgical operations. Another provocative that is worth emphasizing is medicinals such as mercurial diuretics, thiamine hydrochloride, and ergotamine tartrate. Dietary indiscretions have been claimed as provocatives by some, but denied by others.

## TREATMENT

The drug of choice in the treatment of acute gout is colchicine in doses of 0.65 mg. The drug should be given in this amount at the first intimation of pain and should be repeated every two hours until the attack has been greatly ameliorated. Usually 10 to 15 tablets are needed, and they should be given continuously throughout the day and night. If, however, the pain persists, and nausea, vomiting, or diarrhea appear the drug should be discontinued since these may be toxic manifestations of the drug. It may be appropriate to administer small doses of codeine at the onset of the acute attack until the colchicine has had time to become effective. If a course of colchicine fails to bring relief it should be repeated after an interval of two or three days. Acetylsalicylic acid in large doses, 3 to 6 grams a day, may be given in addition to the colchicine therapy, and is frequently of value. Barbiturates are usually necessary to assure sleep. The benefit derived from hot or cold compresses is usually of little moment. Bed rest, the forcing of fluids, and a simple bland diet are all that are needed for the acute attack. It is obvious, of course, that the joint should be moved as little as possible and measures taken to keep the bed clothes off the affected extremity.

In the treatment of asymptomatic gout, by which is meant individuals who have no outward manifestations of gout but who have abnormal uric acid blood levels, two drugs are particularly useful and these are again salicylates and colchicine. Cinchophen and neocinchophen have been widely used but are frequently accompanied by severe toxic and occasionally even fatal results. Usually from 1 to 3 tablets of colchicine a week to 1 to 2 a day is sufficient to keep these people asymptomatic. Although the present concept is that the use of alcoholic beverages and rich foods affects gout very little, clinically it would appear that patients are better off on simple foods. The restriction of foods with high uric content such as brains, pancreas, sardines, is usually practiced.

Rules of temperance usually are sufficient as far as alcoholic beverages are concerned. It is of tremendous importance that an adequate urinary output be maintained.

## SUMMARY

The important features of gout have been discussed and a typical case presented. It is not as infrequent as supposed by many and should be considered in the differential diagnosis of any of the arthritides.

TABLE I

List (1)
Foods which contain a large amount (75 to 1000 mg.) of purine bodies in 100 Gm.:

Sweetbreads
Anchovies
Sardines
Liver (calf, beef)
Kidneys (beef)
Brains
Meat extracts
Gravies
Bacon
Beef
Calf tongue
Carp
Chicken soup
Codfish
Duck
Goose
Halibut
Lentils
Liver sausage
Meat soups
Partridge
Perch
Pheasant
Pigeon
Pike
Pork
Quail
Rabbit
Sheep
Shellfish
Squab
Trout
Turkey
Veal
Venison

List (2)
Foods which contain a moderate amount (up to 75 mg.) of purine bodies in 100 Gm.:

Asparagus
Bluefish
Boullion
Cauliflower
Chicken
Crab
Eel
Finnan haddie
Ham
Herring
Kidney beans
Lima beans
Lobster
Mushrooms
Mutton
Navy beans
Oatmeal
Oysters
Peas
Salmon
Shad
Spinach
Tripe
Tuna fish
Whitefish
Whole grain bread and breadstuffs
Graham bread
Graham crackers
Oatmeal crackers
Rye bread
Rye Krisp
Whole wheat bread
Whole grain cereals
Bemax
Bran
Bran flakes
Cracked wheat
Embo
Graham porridge
Grapenuts
Krumbles
Malt breakfast food
Pep Bran Flakes
Pettijohns
Puffed wheat
Ralston's
Sims
Shredded Wheat
Wheat oats
Wheatsworth
Whole Wheat Krumbles

List (3)
Foods which contain an insignificant amount of purine or no purine:

Beverages:
Carbonated
Chocolate
Cocoa
Coffee
Fruit juices
Postum
Tea

Bread and breadstuffs (except whole grain under list 2)
Butter thins
Corn bread
Corn stick
French bread
Gluten bread
Holland rusk
Soda crackers
Uneeda biscuit
Water rolls
White bread
Zwieback

Others:
Butter
Cheese
Eggs
Fats of all kinds

Fruits of all kinds
Gelatin
Milk—Buttermilk
Malted milk
Condensed milk

Nuts of all kinds
Peanut Butter
Pies (except mincemeat)
Sugar and sweets
Caviar

Cereals (except whole grain under list (2)
Breakfast Brownies
Cornflakes
Cream of Wheat
Farina Post toasties
Grits, Puffed rice
Rice flakes
Rice Krispies
Cornmeal yellow and white

Vitamin Concentrates
Cod liver oil
Halibut oil
Yeast

Vegetables containing an insignificant amount of purine or no purine:

| | | | |
|---|---|---|---|
| Artichokes | Celery | Kohlrabi | Pumpkin |
| Beets | Corn | Lettuce | Sauer kraut |
| Broccoli | Cucumber | Okra | Stringbeans |
| Brussels sprouts | Dandelion greens | Parsnips | Summer squash |
| Cabbage | Eggplant | Potato—Sweet | Swiss chard |
| Carrots | Endive | White | Tomato |
| | | | Turnips |

Vegetable and cream soups:

| | |
|---|---|
| Arrowroot | Sago |
| Hominy | Spaghetti |
| Macaroni | Tapioca |
| Noodles | Vermicelli |

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## ALLERGIC HEMATURIA DUE TO MILK

### REPORT OF A CASE

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NEW ORLEANS

Hematuria associated with anaphylactoid purpura is not new or extremely rare. Allergists encounter such cases fairly often but it is unusual for the urologist to see a patient with hematuria as the only symptom. Osler,[1] in 1914, described 2 cases of Henoch's purpura, one with purpuric skin rash, urticaria, abdominal pain, and persistent hematuria, and the other with purpuric skin rash, colic, and hematuria. Albumin was found in both cases and they were diagnosed as nephritis associated with a skin rash of the erythema group. In the second case almost every urine specimen was smoky and sometimes bright red. Osler stated that months after all signs of cutaneous symptoms have disappeared, hematuria may persist perhaps just enough to tinge the urine.

It was Eyermann[2], however, who demonstrated the association of allergy with the erythema and Schoenlein-Henoch groups of purpura. In 1930, Coca[3] reported 2 cases of "essential" hematuria in individuals of allergic background with associated allergic symptoms. He considered these allergic in origin but this fact was not established. Rhodes,[4] in 1937, reported a case of hematuria from the right kidney due to tetanus antitoxin and Miller,[5] in 1939, reported a case of hematuria from the right kidney due to the ingestion of cod fish. Since then other cases of hematuria associated with allergy, with and without purpura, have been reported. Few, however, were not accompanied by purpura. Among some of the allergens mentioned are tar fumes, antitetanic and antidiphtheritic serums, fava bean, and various foods and inhalants.

The case reported is presented because it is believed that hematuria of allergic etiology, unassociated with purpura, is not frequently seen by urologists. It also serves as a reminder of the possibility of an allergic etiology after all other causes of hematuria have been eliminated, even though

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the preliminary history may not reveal an allergic background.

REPORT OF CASE

J.W.S., a white boy aged 8 years, was first seen February 27, 1945, because of bloody urine of four years' duration. There was no history of trauma, chills or fever, or any illness other than influenza one week prior to appearance of hematuria. The urine varied in color from light pink to a dark brownish smoky color. At times, he passed blood continuously and as a result became weak and dyspneic upon exertion. Frequently, he would have a frontal headache of three to four hours' duration and an occasional burning sensation on micturition associated with the severe bouts of hematuria. For two years he had had occasional pain in the legs, and nine months prior to our seeing him he had presumably had an attack of polyarthritis. He had also had repeated attacks of epistaxis in the past and seemingly the ingestion of eggs frequently produced urticaria. His mother thought the hematuria might have been worse after the ingestion of eggs. The patient drank milk freely with nearly every meal and his mother related that he often consumed as much as a gallon a day! He had been seen by several physicians in his community during the preceding four years and repeated urinalyses had shown many red blood cells as the only abnormal finding.

Physical examination yielded essentially negative findings except for palpable cervical lymph nodes and a harsh mitral systolic murmur poorly transmitted but accentuated in the left lateral decubitus position. The blood pressure was 115/75.

The urine was cloudy and blood tinged in the first and second glasses; specific gravity 1.020, reaction acid, faint trace of albumin, no sugar. Microscopic examination showed innumerable red blood cells and 4 to 6 white blood cells per high power field. A stained smear of the sediment showed no pus or organisms. The phenolsulfonphthalein kidney function test was 55 per cent in one hour and ten minutes. Excretory urograms were normal. The red blood count was 4,000,000; white blood count 9050; hemoglobin 10.3 Gm.; eosinophils 5 per cent, neutrophils 55 per cent, and lymphocytes 40 per cent. Serological tests for syphilis were negative.

The child was hospitalized for additional study. Further urinalyses showed the urine still loaded with red blood cells; the specific gravity was 1.011; a trace of albumin; no sugar. A urine culture was negative. The nonprotein nitrogen was 37.5 mgm. per cent; carbon dioxide combining power 49.4 vol. per cent; dextrose 87; prothrombin time 100 per cent; platelet count 537,500; and sedimentation rate 26 mm. The Rumpel-Leede phenomenon was not demonstrable. Roentgenograms of the heart and lungs and an electrocardiogram were within normal limits.

Cystoscopy was performed under general anesthesia. The bladder and urethra were normal and bloody urine was seen spurting from both ureteral orifices. After these studies the parents, who lived out of town, desired to take the child home. They agreed, however, to return several weeks later for further study, especially allergy tests and reexamination of the heart.

The boy returned on June 26, 1945, with continuation of the hematuria. He had had no urticaria during the interim and appeared healthy. The urine showed many red blood cells; innumerable amorphous urate crystals; four plus hemoglobin; and a trace of albumin. Blood urea nitrogen was 16 mg. per cent. Blood studies showed a red blood count of 4,500,000; hemoglobin 13 Gm.; white blood count 15,000; eosinophils 9 per cent; neutrophils 54 per cent; lymphocytes 35 per cent; mononuclears 2 per cent; and a sedimentation rate of 13. Blood pressure was 102/64. Roentgenograms of the lungs and heart and an electrocardiogram were within normal limits. Skin tests for food allergens gave negative results.

The child was seen by Dr. Vincent J. Derbes in the section of Allergy and it was then decided to try to determine by means of an elimination diet whether or not the hematuria was due to an offending allergen. The patient was allowed to return home. The first diet to be followed was the Rowe No. 4 consisting of milk and tapioca. Because tapioca was not procurable, the first diet consisted only of milk. The mother wrote that the first three days of the milk diet greatly increased the amount of hematuria; she reported that the patient urinated "almost pure blood." His mother was then instructed to eliminate milk and all milk products from his diet.

The patient was next seen about nine months later on April 2, 1946. His mother stated that on discontinuance of the milk diet the urine gradually regained a normal appearance. When milk was eliminated from his diet, his attacks of hematuria occurred only every two to three weeks and lasted only several days, the urine being colored a faint pink; whereas when milk was included, hematuria was almost continuous and the urine was much darker in color. The child had had no fever, pain, edema or rash at any time since last seen. He was again hospitalized and put on a milk free diet.

At the time of admission the urine was normal. The blood pressure was 106/70. Although the mitral systolic murmur was still present, no evidence of carditis existed. Skin tests for food allergens were repeated and were negative.

The boy was taken out of the hospital one morning and that afternoon put on a milk diet. Whereas urine obtained that morning was clear and showed no red blood cells microscopically, before he retired that night the urine had become a brownish color. The next day a voided specimen was dark brown. The urine showed a positive reaction for

occult blood and a slight trace of albumin. Microscopic examination revealed a moderate number of red blood cells. The urine appeared to contain about 2 to 3 plus hemoglobin. The child had experienced no pain, rash, or other discomfort with the episode. The patient then went home with instructions to take no further milk.

On September 10, 1946, the child's mother said that the patient had had no hematuria unless he had received unboiled milk from outside sources. She had discovered that the child would not have hematuria if he drank boiled milk but hematuria invariably followed the ingestion of unboiled milk. This mechanism has been explained by Ratner[6] as being due to the denaturation of the whey proteins by intensive heat with the resulting loss of their antigenic properties. The lactalbumin is coagulated by heat and therefore is not readily absorbed through the gastrointestinal tract.

A recent communication from the child's parents revealed that he drowned accidentally June 25, 1947. Until that time he had had no further hematuria, epistaxis, or pain in the legs or joints. He played hard, never became fatigued and had gained considerable weight. He had continued to drink boiled milk and only occasionally did a mild nettle rash appear.

SUMMARY

A case of allergic hematuria produced by the ingestion of unboiled milk in an 8-year-old boy is presented. Although reactions to skin tests were repeatedly negative, ingestion of unboiled milk produced hematuria and its withdrawal or the boiling of milk eliminated it. Appearance of the urine varied from pink to red and at times had a brownish color with both red blood cells and hemoglobin present. Hematuria was the only presenting symptom. A history of urticaria, epistaxis, headache and one attack of polyarthritis, but no purpura, was obtained. In retrospect, the epistaxis may have been a purpuric manifestation. The fact that while at home on a milk free diet he had occasional, transient periods of hematuria is probably due to the fact that it is extremely difficult to eliminate unboiled milk entirely from the diet.

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# OBSTRUCTIVE ESOPHAGEAL LESIONS IN GASTROINTESTINAL PRACTICE*

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NEW ORLEANS

Obstructive esophageal lesions in gastrointestinal practice among adults vary from the extreme of hysterical dysphagia to the organic lumen obliteration of neoplasia. It is dysphagia that initiates the etiologic search; meticulous survey of the swallowing derangement and related symptomatology aid in preliminary diagnostic speculation.

INCIDENCE

In a decade review of gastrointestinal studies, dsyphagia, present in 852 instances, occurred in 8.2 per cent of patients. In this series esophagitis of acute and chronic systemic disease, and that related to ingested chemicals, intentional or accidental, was excluded.

Of the 852 patients, 639 (75 per cent) were of purely functional origin. Intraesophageal disease, including roentgen demonstrable esophagospasm, was present in 170 (20 per cent). Lumen compression from extraesophageal disease was encountered in 34 (4 per cent). Deglutination derangement of neurologic origin was present in 9 patients (1 per cent).

Pertinent to this presentation are 213 cases of actual esophageal obstruction, which roughly represent 25 per cent of those studied for dysphagia. The conclusion that 75 per cent of dysphagics are functional individuals would, if not evaluated, seemingly indicate dysphagia to be

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overwhelmingly a hysteroid manifestation of minimal clinical importance.

With the presumption that esophageal obstruction, secondary to pressure from without and neurologically originating derangements of deglutination, have relation to this discussion only in the established importance of differential diagnosis, we are left with 170 cases of intraesophageal obstructive disease. Herein are included 94 (55.3 per cent) cases of esophagospasm and 34 (20 per cent) instances of neoplasm. Diverticula, 3 pharyngoesophageal, 2 midesophageal and 6 cardiac, represented 6.5 per cent of this group of dysphagics. Benign stricture (11.8 per cent) of undetermined etiology presented a problem in 12 instances; in 5 additional cases it was associated with a congenitally short esophagus; in 3 it was a part of scleroderma. There were 4 individuals with severe varices (2.4 per cent) which, with associated esophagitis, excited dysphagia. Three foreign body impactions (1.7 per cent) proved obstructive. We had only 2 proved instances of peptic ulceration of the esophagus (1.2 per cent) which, with related spasm, were obstructive. Syphilitic stricture and Boeck's sarcoid involving the esophageal wall, were rarities encountered, one of each.

### DIAGNOSIS

Esophagoscopy, in our experience is primarily a diagnostic procedure. Differential diagnosis without confirmatory observation is rarely acceptable, because of the frequency of concomitant disease. Pathologic mucous membrane is taken for biopsy; locally applied filbrin foam seals the site, controls bleeding, and possibly further minimizes the danger of mediastinitis. In this series all neoplasias were biopsy confirmed squamous cell carcinomas; 18 additional biopsies revealed non-neoplastic tissue. Therapeutically, 4 instances of stricture and 3 of cardiospasm necessitated initial per-esophageal bouginage; the 3 foreign bodies (meat impactions) were removed through the scope; in one patient with varices these were sclerosed with sodium morrhuate.

Roentgen demonstration of the various esophageal lesions responsible for dysphagia would call for too extensive illustration for this type of presentation. Fluoroscopic study of esophageal function is essential, since many conditions producing dysphagia, such as paralysis, muscular weakness, or incoordination, do not produce mucosal changes. The differentiation is not as simple as often considered, but may often necessitate combined roentgenologic, endoscopic, and biopsy study to conclude that the lesion results from disruption of normal physiology. Templeton in his classic on x-ray examination of the stomach includes a thorough evaluation of roentgen esophageal study; in addition to thoroughly presented routine observation, he adds his observations on primary and tertiary peristaltic waves in relation to cardiospam and other esophageal disease.

### TREATMENT

Nonoperative management of obstructive esophageal disease is restricted to general measures to improve the mental and physical well being of the patient, dietary measures, vitamins, applicable antispasmodics, analgesics, physiotherapy, deep roentgen therapy, and bougienage.

The principal dietary instruction should be the outlining of a diet compatible with the patient's lesion in an effort to maintain oral nutrition at as near normal requirements as is possible. Excessive heat or cold, condiments, alcohol, and mucosal irritants are contraindicated. The dietary management of peptic esophagitis is that of an ulcer elsewhere. Tube feeding may be indicated in an effort to gain nutritional benefit, to maintain a lumen, and to rest the esophagus.

Amyl nitrite, nitroglycerine and aminophyllin are often effective in the relief of cardiospasm, reflex esophagospasm, and spasm associated with Plummer-Vinson syndrome. This is our confirmed observation in proved cases, but since it is contrary to the opinion of such authorities as Bockus, Ritvo and McDonald, and others, we advocate careful diagnostic study in all instances since these same drugs are effective in coronary and cholecystic spasm. The other so-called antispasmodics, including trasen-

tine, syntropan, pavatrine, trocinate, profenil, octin, Abbott AP 43, have, in our clinical, roentgenologic, and bougie control series, been entirely ineffective both orally and parenterally. Atropine and its derivatives are actually contraindicated. Prostigmine and depropanex have no esophageal relaxing value.

Analgesics and sedatives have their place. Habit forming drugs are limited to emergency usage.

Vitamins have limited applicability when malnutrition has produced deficiency. Etzel would have us give thiamin as a specific measure.

Acid neutralizers relieve the pyrosis of esophagitis, especially when gastric regurgitation is the excitant.

Hyperpyrexia, local diathermy, and spinal anesthesia have poorly confirmed and largely debatable therapeutic worth in any form of esophageal disease.

The sclerosing of varices is of confirmed value.

Deep x-ray and radium therapy of esophageal neoplasms may be more than symptomatically ameliorative in lymphosarcomas, but is generally felt to be only that effective in carcinomas.

Bougienage has been practiced for over 50 years in relieving dysphagia. Passive dilatation is applicable to all cases of esophageal narrowing; active divulsion is restricted to instances of spasm and has its greatest efficiency in cardiospasm.

Steel shaft fiber bougies and mechanical divulsors used through the esophagoscope, necessarily involve a semioperative procedure; they are applicable to the preliminary management of cases in which scarring or fibrous stricturing exists. Sippy's graduated olives and those of others in various sizes and shapes attached to a fiber and guided by a swallowed thread, are introduced fearlessly by persistent adherents. Unguided semistiff woven fiber bougies in competent hands are excellent adjuncts.

Hurst introduced his mercury filled bougie in 1913. These, carried and guided through the esophagus by their weight, open a passageway by their size. They are easily passed, cause little discomfort and are unquestionably the safest dilators available. Successively larger tubes (21 to 40 English) are passed at a single sitting. In most cases the largest meets with only slightly more resistance than the smallest in spastic disease.

Additional favorable factors in the use of Hurst bougies include: (1) In the instance of narrowing at several sites, all are dilated to the same extent by one passage. (2) Patients may be taught self bougienage. (3) No fatality has been recorded from their use. These bougies have limitations, are more satisfactory when the cardiac opening is dependent without too great sacculation, the larger sizes often prove annoying to patients because of the bulk in the pharynx.

The history of bag divulsion dates back to J. C. Russel's work of 1898. In 1906, Plummer modified Russel's instrument and introduced into prominence the rubber covered silk balloon hydrostatic pressure. Moersch and Vinson, with their excellent work and statistic accumulation, have sustained their preceptor's teachings. This instrument is a rubber tube supported by a whalebone staff tipped with an olive, just behind which is a rubber covered silk balloon which is dilated by hydrostatic pressure regulated by special adaptors. Introduction is carried out over a previously swallowed silk thread.

In 1929, Frank Smithies designed the pneumatic dilator, a modification of Plummer's apparatus. This dilator, being portable, requiring no special water supply, being devoid of whalebone staff, but having a metal shaft with safety control, and being of inexpensive construction, was a notable achievement. It rendered fluoroscopic control of divulsion dilatation practical. Its only essential distinction from the Plummer dilator, however, is that it is pneumatic.

Since September 1938, we have used a mercury pneumatic dilator. This dilator and divulsor combines the principles of Hurst, Plummer and Smithies and achieves a position of maximal efficiency and safety.

It is a No. 21 Hurst mercury tube, on the distal end of which, 7.5 cm. from the tip has been incorporated a rubber covered silk bag, 11 cm. length, to which a small catheter runs through the mercury filled tube. By this means distention, under the control of a manometer, may be effected.

The distinct advantages of this instrument are that: (1) A swallowed guiding thread is not required; it is easily passed, being the size of the small Hurst dilator. It is carried through by virtue of its contained 21 ounces of mercury sufficient to force the closed sphincter, but not weighty enough to traumatize or perforate the esophagus. (2) This type dilator is more easily passed with less discomfort than any other type, and shows less tendency to coil in a dilated prediaphragmatic esophagus due to the tendency toward rigidity in the distal six inches. (3) Only a single instrumentation is required, as contrasted to the passing of an olive prior to the use of a Plummer dilator and the use of graduated Hurst bougies. (4) It offers controlled pneumatic divulsion, which is more practical and safer than hydrostatic dilation. (5) It is applicable to fluoroscopic control.

Dilatation and divulsion rarely fail in cardiospasm or benign stricture, and the prognosis is such that one seldom considers operative procedures. Gastrostomy is merely palliative. Retrograde dilation with Tucker's instrument and divulsion manually, or by forceps from the gastric side, are major procedures reserved for those few instances in which adequate indications exist.

The surgery of dysphagia includes diverticulum resection especially applicable to the pharyngo-esophageal variety, extrathoracic esophageal construction in complete lumen obliteration, esophagogastric plasty in some instances of cardiospasm and the esophageal resection for malignancy. Torek's achievement of a five year survival after thoracic esophagectomy for carcinoma has not been duplicated.

From the gastroenterologists' aspects surgical management is a procedure elected only when urgently indicated, extremely dangerous when the thoracic esophagus is involved and rarely produces a result freeing the patient from the need of postsurgical endoscopy and dilatation.

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## THE SHORTCOMINGS OF CERTAIN OPERATIVE PROCEDURES IN THE TREATMENT OF PULMONARY TUBERCULOSIS*

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One might also title this presentation, "Problems Encountered in the Selection of Operations for Patients with Pulmonary Tuberculosis." What is meant more precisely is that most of the surgery for pulmonary tuberculosis is pretty well standardized today, but there are times when operations fail to accomplish what was intended of them. This is true in other fields of surgery just as much as it is in the chest. For example, gastroenterostomy is an appropriate operation for the treatment of certain peptic ulcers, but it will fail in a certain number of patients. This does not mean that anything is wrong with the gastroenterostomy or its application in any particular patient. The same is true with lesions of the lungs. Thoracoplasty has been and continues to be a most effective operation for closing tuberculous cavitation in a lung, but it will fail in this mission in a number of patients. It is most disconcerting to have a patient upon whom we have performed thoracoplasty successfully and with apparent cavity closure return to us or to another thoracic surgeon in a few years with the cavity reopened.

### TENSION CAVITIES

This is the first problem we shall consider. When a patient has large cavitation, we assume it to be of the tension type. This assumption is not without foundation. These patients, to be sure, must first be bronchoscoped to determine the nature of the bronchus leading to their cavitation. If

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we find the bronchus normal, we may proceed to treat this patient in the conventional manner, and that is, of course, by doing a conventional thoracoplasty. One cannot always be sure that the bronchus is normal from purely bronchoscopic findings. Many of these cases have bronchostenosis around the corner where it cannot be demonstrated by endoscopy. We, therefore, also subject these patients to manometric studies of the cavity pressure itself. Naturlaly, increased bronchial pressure, as evidenced by needling such a cavity, means endobronchial pathology. Such a cavity will not close by ordinary surgical methods.

If by the above bronchoscopic findings and manometric studies we find the bronchus to a cavity to be normal, one may expect to get cavity closure with a conventional thoracoplasty. We cannot take our results for granted and must not rely solely on plain roentgenograms as they are not sufficiently convincing. One must depend on continued negative cultures by bronchial lavage and additional bronchiographic evidence that the lesion is closed. If, at the time of such investigation, one finds a cavity remaining, a problem then ensues as to further management. Although we shall not take time to discuss treatment of this type of case, further surgery resolves itself in either a revision thoracoplasty (the results of which have been generally disappointing), open cavity drainage, or lobectomy. The selection of these subsequent procedures entails a consideration of many other factors, one of which deals with how much disease the patient might have had in other portions of the lung.

If in our endoscopic studies one finds a bronchus to be diseased, it behooves one to treat that bronchus primarily before subjecting the patient to collapse. The treatment of such endobronchial disease is a subject in itself and may resolve into a preliminary course of streptomycin by nebulization or to actual cauterization of the ulcer itself. If, after such treatment, the active phase of bronchial tuberculosis has been somewhat controlled, we find evidences of a trap valve action on the cavity, we must put a safety valve into the cavity before attempting surgical collapse. To accomplish this, we institute a Monaldi cavity drainage operation preliminary to the actual thoracoplasty. Monaldi drainage is never done after the thoracoplasty unless it was an omission on our part. Since any form of actual cavity drainage necessarily infects the surrounding field, such a drainage should be done in an area which is not accessible to the main thoracoplasty. In order to effectually release this region in the ultimate collapse, rib resection is done in this area before inserting a catheter in the cavity. The preferable location for such a procedure is, of course, anteriorily and the anterior thoracoplasty of Haight has been our choice, performing it some ten days before the Monaldi. Monaldi drainages are now supplemented by suction drainage to further effect a reduction in the size of the cavity. Such suction is maintained through the intervals during which the posterior thoracoplasty is eventually accomplished. Needless to say, the follow-up posterior thoracoplasty must be thorough in that complete rib lengths are taken, but the field of the Monaldi must not be entered. Since this area has been previously released, the cavity should be entirely free, both by the rib resection and a thorough pleurolysis. The tube can eventually be removed when bronchial closure is insured, and oftentimes one may find that the tube extrudes of its own accord after a number of weeks. We must emphasize here that the Monaldi operation by itself is almost worthless, and most internists and surgeons are now in accord that it be followed by an extensive thoracoplasty.

Other examples of tension cavity may be found in so-called pneumothorax failures. The pneumothorax in such a case may be virtually total, with the exception of the cavity itself. Needless to say, such a pneumothorax is not only worthless but deleterious inasmuch as it collapses only the good portion of the lung. Such patients frequently do poorly clinically, as evidenced by loss of appetite with continued weight loss, continued febrile bouts, both from cavity block-

age and from effusion formation, and poorly mentally from continued reports of a sputum which is positive for acid-fast bacilli. The simple expedient of reexpansion of the pneumothorax, even though the cavity still remains patent, often demonstrates an about-face on their clinical well-being and they again begin to gain weight and become afebrile. The ultimate handling of the cavity is no different than in the preceding types, and eventually resolves itself into thoracoplasty with or without a complementary Monaldi drainage.

Of passing interest is an example of tension cavity which we formerly subjected to a so-called plication procedure. We have only utilized a plication operation twice, and I must confess that we no longer use it. It was used in one case where the old type Brauer operation was performed. This type of thoracoplasty was far from successful in accomplishing satisfactory cavity closure for this patient. During the revision thoracoplasty, the pleura is entered and the lung freed from surrounding parietal structures. This is effected through a trap door incision through the upper four or five ribs posteriorly. Intercostal bundles are salvaged as they are later used in the closure. The region of the cavity is then folded manually over itself and thus plicated by at least two rows of sutures, much as one does in an intestinal inversion. The intercostal bundles, now longer, due to resected corresponding ribs, are scrolled and sutured back in place and the wound closed in the conventional manner. Although statistics are not readily available, such plication procedures have been rarely used in this country. They are technically difficult and a successful plication is hindered considerably by the induration of the diseased lobe. From the tension standpoint, one would not always expect to effect cavity closure from information available from our present knowledge. Such procedures have been largely supplanted by lobectomy which is technically easier and far more effectual.

### TUBERCULOUS EMPYEMA

The patient with tuberculous empyema has been a problem for many years. Reports still reach the literature with attendant high mortality for surgical collapse in such cases. The reason for this mortality is hard to comprehend. We believe that patients with tuberculous empyema, properly managed, will carry little if any greater mortality than the average thoracoplasty. Much stress, however, lies in the preparation of that patient for such surgical collapse. This entails rigorous medical management, however, not prolonged, regulating protein intake and insuring vitamin, caloric, and fluid balance. During this period it becomes necessary to consider the empyema itself, and many clinicians have resorted to frequent aspirations to prevent absorption of the tuberculous pus. Nothing is more disappointing to a thoracic surgeon than the finding of multiple sinuses in the chest wall from such repeated needlings, frequently located in the region where the incision for the thoracoplasty is contemplated. We agree that tuberculous pus should be removed, but we prefer to remove it completely and continuously by the simple expedient of intercostal drainage by small catheter. Such catheter drainage will not result in mixed infection if connected to an underwater trap. Moreover, the catheter may be placed low anteriorly where its site will not infect the subsequent thoracoplasty. Such drainage by catheter is not only innocuous but better prepares the patient by eliminating febrile bouts that ordinarily accompany an empyema. Moreover, it is a great boon mentally to these patients to at least find out they can feel and eat better even before the start of major collapsing procedures. Streptomycin is a necessary adjunct both parenterally and within the pleural cavity. It aids in reducing the number of bacilli in the pleural wall. One then proceeds with a conventional thoracoplasty, removing long rib lengths to include the first two or three cartilages and all transverse processes. Continuous suction is maintained on the empyema drainage as it

is mandatory to obliterate every iota of pleural pocketing in such a case.

### BILATERAL TUBERCULOSIS

In bilateral disease where thoracoplasty or permanent collapse is necessary on one side, we at once concern ourselves with the better side. Any such contralateral lesions which do not clear themselves promptly on bed rest must be considered for pneumothorax. Thus a temporary collapse is instituted for the better side preliminary to the surgical collapse on the worst side. Not infrequently, we become confronted with a pneumothorax failure on the better side. We must then resort to some other temporary collapsing measure, with or without streptomycin, to prepare the patient for major surgery. Consequently, one must consider adopting an extrapleural separation or possibly a pneumoperitoneum to fulfill this purpose. We are trying to give this patient one good leg to stand on before we tackle his worst lung. In so doing, postoperative spread is obviated which can be no little concern in managing these patients. Pneumoperitoneum is being used more frequently of late with gratifying results. Phrenic interruption is not used as it limits the motion of the diaphragm too seriously in a patient who has been operated upon. On one score, pneumoperitoneum is less hazardous than pneumothorax in that effusions do not usually occur. Needless to say, an effusion complicating a pneumothorax on the patient's better side can be a real problem. We lay down one maxim in utilizing temporary collapse for the better lung: it must be curable by temporary measures. It, therefore, must possess no large cavitation because permanent collapse would be thus necessary here as well. One can, however, do limited bilateral thoracoplasties. Vital capacity and spirometric determinations are a "must" in many of these. There is a certain limitation to the amount of bilateral disease any one person can have and still undergo collapsing procedures. This limitation is even more pronounced when the type of collapse is permanent. In one of our patients who was hemorrhaging at the time of admission, it was assumed that the hemoptysis arose from his worst lung. To obviate a critical flare-up, a contralateral pneumothorax was established as we thought he had too much disease to undergo thoracoplasty on the worst side. As frequently happens with the establishment of the contralateral pneumothorax, not only did the lung clear but some of the disease on the worst side diminished. He was 68 years old incidentally, but went through his surgery without incident and ended up with repeated negative cultures. To give some interesting statistics on this type of patient, I wish to quote the results of Buxton of Ann Arbor who reported 44 bilateral cases handled thus with 83 per cent sputum conversions. There are other sizeable series: O'Brien and Tuttle reported 85 such cases with comparable results. This consideration of bilateral disease is fairly brief as the subject is huge, but I hope you will bear with me.

### CASES WITH HEMORRHAGE (X-RAY NEGATIVE)

One of the big problems that confronts us not too infrequently is the patient who shows a fairly normal roentgenogram but continues to have hemoptysis. Acid fast bacilli may or may not be demonstrable in their sputa and this adds to the concern. The first rule, of course, is to bronchoscope these patients. This may show nothing, but if an ulcer is seen, the diagnosis may be made. Those who have a normal endoscopic appearance should be subjected to total bronchograms, as most frequently the site of the hemorrhage and sputum can then be found. The usual finding is bronchiectasis, either under the old thoracoplasty, or in another portion of the lung previously unsuspected.

### EXTRAPLEURAL PNEUMONOLYSIS

I should like to mention a few things about extrapleural separation. This was brought to our attention earlier in this presentation in bilateral cases where intrapleural pneumothorax was not successful. When the operation was first introduced some years ago, we did not know into what the indications would eventually resolve themselves, or the outcome of the procedure. Now we emphasize that the lesions

treated by such an operation must be temporary in nature, or curable by temporary means. It may be used as a preliminary operation when another more permanent type of collapse is anticipated. Usually in the latter case, the pneumonolysis is followed by a thoracoplasty. It can either be done anteriorly or posteriorly, with or without rib removal, or even replacing the rib. The indications for such a procedure, of course, must be necessarily limited as one could ordinarily perform such a thoracoplasty primarily. One of the chief indications for this operation is the control of hemorrhage where conventional pneumothorax cannot be done. It produces instant collapse of the bleeding portion of the lung and affords a better risk patient for the subsequent thoracoplasty. Naturally, we cannot do the thoracoplasty to stop the bleeding, since it is a staged procedure and the patient may bleed to death before a sufficient number of stages are completed.

Formerly some of these pneumonolyses were followed by instillation of a foreign substance to make them permanent. Paraffin was one of these, but muscle, fat, oil, and special gauzes have been used. It is well to point out the hazards of paraffin pneumonolyses, particularly when a large amount of paraffin is used. In the first place, it is a foreign body which may provoke an extrapleural effusion. The space thus formed may be the source of a chronic infection. Again, the paraffin frequently shifts its position and can no longer be trusted as an effective plomage agent for the underlying disease. These patients will probably come to eventual thoracoplasty, which in retrospect, one could have done primarily. Small paraffin fillings in selected cases sometimes continue to do well, but the agent is not entirely innocuous of foreign body reaction, and therefore, not ideal. Recently, plastic materials have been used but conclusions cannot yet be drawn.

The defeat of extrapleural pneumothorax, or filling the space with air refills, lay in the inability to reexpand many of these lungs. Such an extrapleural space when maintained over a period of years, must eventually become the seat of chronic infection and a thoracoplasty eventually follows. The rib removal in such a case must be necessarily delayed until all signs of infection have been controlled.

### CONCLUSIONS

There are a number of additional problems which one encounters in this field which have not yet been considered due to the limitation of time. One of these is the consideration of primary lobectomy for the treatment of tuberculosis of the lungs. Pulmonary resection has a promising outlook in carefully selected patients. The subject is complex and quite large, consequently we cannot give it due respect today. Then there is the problem of the tuberculoma, basal tuberculosis, the proper utilizing of phrenic nerve interruption, and extensive intrapleural pneumonolysis.

In summing up, let us merely state that results from the surgery of tuberculosis of the lungs can be quite gratifying if one studies the patient from all available sources of investigation. A properly applied operation will therefore give good results when it is known what is to be expected of the procedure.

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## PROLAPSE OF GASTRIC MUCOSA INTO THE DUODENUM*

### CASE REPORT AND ANALYSIS OF REPORTED CASES

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A cursory review of the literature pertinent to the prolapse of gastric mucosa into the duodenum reveals a varied attitude toward this condition and numerous methods of treatment. Many clinicians and roentgenologists have believed this to be a mere curiosity often seen incidental to the routine barium examination of the gastrointestinal tract and not the primary pathologic lesion occasioning the examination.

An increasing body of evidence, however, tends to show that this is not the case

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and that prolapsed gastric mucosa may give rise to a symptom complex which may be disabling to the patient and for which a definite course of treatment is indicated. An analysis of the literature discloses a total of 33 cases of prolapsed gastric mucosa proved at operation, to which we have added the 34th. The first case reported by Von Schmiden[1] in 1911 was followed in rapid succession by others. (Table I). Four additional cases attributed to French[3] and 6 other cases reported by Ira A. Ferguson, but not published, and therefore not available for analysis, bring the total to date to 44 cases. Over 100 cases are reported as noted on x-rays but not confirmed by surgery, and therefore, are not included in this report.

This condition has been found and proved by surgery in only 1 case in Charity Hospital at New Orleans in over 500,000 admissions since 1937. We feel that this does not represent the true incidence of this condition, and that it may be a common cause of epigastric distress and a potential source of obscure gastrointestinal tract bleeding overlooked by the radiologist or ignored by the clinician.

Scott,[11] who has done rather extensive investigative work on this problem, feels that this condition is more than a mere roentgenographic varient and should be considered a clinical entity. However, considerable controversy still exists. How much importance should be attached to this condition remains unanswered. Bockus[17] believes that only severe prolapses associated with gastritis can cause symptoms and then only after producing partial pyloric obstruction. Alvarez states that the roentgenological appearance of prolapsed gastric mucosa has no significance and does not explain the symptoms. Impetus for further consideration of this condition is provided by A. Melamed[4] of Milwaukee, who demonstrated 25 cases so diagnosed in which the patients were relieved of symptoms following medical or surgical care. Others have pointed out the dangers of overlooking some associated pathology, e.g., ulcers, tumors, and carcinomas.

The roentgen signs produced by this condition were first described by Eliason, Pendergass, and Wright in 1926.[15] Feldman[18, 11, 5] and others report an incidence of redundant gastric mucosa in approximately 0.1 per cent of all gastrointestinal cases, and which is not recognized as such until the mucosa prolapses into the pyloric canal, and presents a characteristic mushroom appearance in the first portion of the duodenum. Scott[11] feels that prolapses are overlooked for three reasons: (1) The examiner is not thinking of them; (2) filling defects produced are confused with ulcer or duodenitis; and (3) when recognized, no clinical importance is attached to them.

At operation[2] the prolapsed portion of gastric mucosa appears like a loose collar of redundant hypertrophied mucosa. The prolapsed folds may appear as normal mucosa, soft and pliable, and not fixed, or the mucosa may be thickened as in chronic gastritis. Studies of the normal gastric antrum reveal the mucosa and muscularis to be connected by soft, yielding submucosa composed of loose areolar tissue containing blood vessels, lymphatics, and nerves. Scott, in studying over 100 autopsy specimens, demonstrated the mucosa to be movable over a distance of 1 to 1½ inches. Normal mobility of this range is apparently not enough to permit a prolapse.

Studies of associated disturbed physiology of the stomach, as determined by gastric acidity, fail to show any correlation. Gastric acidity from O Free and ± 13 total, to 75 free and 90 total is reported. Gastric motility as demonstrated under fluoroscopy with a barium meal also varied with the degree of pyloric obstruction present.

The history of symptoms ranged from several weeks to ten or more years and from mild to severe. The more consistent complaints were those of epigastric distress aggravated by taking of food, especially solids, epigastric fullness, and in severe prolapses nausea and vomiting, probably indicating varying degrees of pyloric obstruction. A symptom reported in several cases,[10, 12] and the primary complaint in our

TABLE I

| Author | Year | Age | Sex | Symptoms | Duration | Hemorrhage | X-Ray | Acidity | Operation | Pathology | Result |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Von Schmieden | 1911 | 27 | F | Vomiting and epigastric pain | 7 Months | None | Irregularity of pylorus | Normal | Excision of mucosa | Fold of mucosa inflamed | "Cure" |
| Eliason, and Wright | 1925 | 56 | M | Not stated | Not stated | None | Prolapsed mucosa | Not stated | Gastrostomy | Prolapsed mucosa | Not stated |
| Eliason, Pendergass, and Wright | 1926 | 61 | M | Epigastric cramps | 5 months | None | Constant defect of duodenum | Not stated | Gastroenterostomy, excision of prolapsed mucosa | Prolapsed mucosa | No follow-up |
| Eliason, Pendergass, and Wright | 1926 | 29 | M | Indigestion | Not stated | None | Constant defect of duodenum | Not stated | Gastroenterostomy, excision of prolapsed mucosa | Prolapsed mucosa | "Healed" |
| Meyer and Singer | 1931 | 42 | M | Indigestion and vomiting | 2 months | None | Irregularity of pyloric area | 0 Free | Resection mucosa | Chronic gastritis | Well 3 months later |
| Pendergass and Andrews | 1935 | 70 | M | Indigestion | 15 years | None | Prolapsed mucosa | Normal | Excision mucosa, gastroenterostomy | Redundant mucosa, Ulcer post. wall | Improved, no follow-up |
| Pendergass and Andrews | 1935 | 37 | F | Indigestion | 1 year | None | Prolapsed mucosa | 30 Free 86 Total | Excision of mucosa | Prolapsed mucosa | Well 4 years later |
| Pendergass and Andrews | 1937 | 46 | M | Indigestion | 5 years | Two bouts hematemesis | Filling defect pylorus | 45 Free 60 Total | Pyloroplasty | Redundant mucosa | Recurrence symptoms 2 years |
| Rees | 1937 | 53 | F | R.U.Q. pain, fullness | 5 years | None | Mucosal pouching 6 hr. residue | Not reported | Closure pylorus, Gastroenterostomy | Prolapse mucosa | Unimproved |
| Rees | 1937 | 43 | F | R.U.Q. pain, fullness | Years | None | Prolapse. No retention | Not stated | Section pylorus, excision mucosa | Prolapse mucosa | Well. Follow-up not stated |
| Rees | 1937 | 33 | M | Vomiting | 3 months | None | Mucosal prolapse | Not stated | Excision mucosa, | Prolapse mucosa | Well. Follow-up not stated |
| Boher and Copleman | 1937 | 57 | M | Pain, vomiting hematemesis | 20 years | Hematemesis 15 years | Prolapse. No retention | Not stated | Excision mucosa, Horsley plyoroplasty | Prolapse mucosa | Died 4 months, septicemia |
| Archer and Cooper | 1939 | 29 | F | Indigestion, epigastric distress | 11 years | Hematemesis, melena, anemia | Prolapse mucosa | 32 Free 42 Total | Pyloroplasty | Prolapse, tight pylorus | Relieved when discharged |
| Archer and Cooper | 1939 | 27 | M | Epigastric pain, vomiting | Not stated | None | Prolapse mucosa | Not stated | Heineke-Mikulicz pyloroplasty | Prolapse mucosa | Relieved when discharged |
| Archer and Cooper | 1939 | 51 | M | Tarry stools, weakness, anemia | 2 years | Melena | Negative | Not stated | Pyloroplasty | Prolapse, tight mucosa | Recurrence of hemorrhages |
| Archer and Cooper | 1939 | 58 | M | Indigestion hemorrhages | 3 years | Hematemesis, melena | Prolapse. Old gastroenterostomy | Not stated | Horsley pyloroplasty | Healed ulcer, prolapse | Small hemorrhage 6 months later |
| Melamed and Hiller | 1943 | 39 | F | Fainting, weakness | 3 months | Melena | Filling defect antrum | Not stated | Excision mucosa | Hypertrophy, healed ulcers | Well 1 month later |
| Norgore and Shuler | 1945 | 61 | F | Epigastric pain | 1 year | None | Prolapse mucosa | 75 Free 90 Total | Gastric resection | Chronic gastritis, prolapse | Died 4 months Post Surgical normal |

| Author | Year | Age | Sex | Symptoms | Duration | Hemorrhage | X-Ray | Acidity | Operation | Pathology | Result |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Nargure and Shuler | 1945 | 42 | M | Epigastric pain | 2 years | None | Healed ulcer, prolapse | 69 Free 85 Total | Gastric resection | Gastritis, prolapse with obstruction | Well at discharge |
| Scott | 1946 | 25 | M | Indigestion, epigastric pain | 19 months | Occult blood | Deformed bulb | 11 Free 27 Total | Resection mucosa Finney pyloroplasty | Normal mucosa | Well 1 year later |
| Scott | 1946 | 41 | M | Epigastric pain | 2 years | Occult blood | Prolapse mucosa | 0 Free 13 Total | Resection mucosa, Heineke-Mikulicz pyloroplasty | Prolapsed mucosa | Well 1 year later |
| Scott | 1946 | 34 | M | Indigestion, epigastric pain | 2 years | Occult blood | Protruding mucosa | Normal | Resection mucosa, Heineke-Mikulicz pyloroplasty | Prolapsed mucosa | Well 1 year later |
| Scott | 1946 | 31 | M | Epigastric pain | 5 months | Hematemesis 500 cc. | Prolopse mucosa | 34 Free | Resection mucosa, Heineke-Mikulicz pyloroplasty | Prolapsed mucosa | Well 6 months later |
| Mackenzie, MacLeod, Bouchard | 1946 | 29 | M | Fullness, Indigestion | 20 months | None | Filling defect | Normal | Resection mucosa, Heineke-Mikulicz pyloroplasty | Prolapsed mucosa | No follow-up |
| Mackenzie, MacLeod, Bouchard | 1946 | 48 | M | Epigastric pain, ulcer healed | Not stated | None | Narrowing prepyloric region | 0 Free 30 Total | Resection mucosa, Heineke-Mikulicz Pyloroplasty | Simple inflammation | No follow-up |
| Appleby | 1947 | 64 | F | Weight loss, indigestion | 10 years | Anemia, occult blood | Prolapse | 0 Free 56 Total | Gastric resection | Gastritis, superficial ulcer | Well until dead 3 years later |
| Appleby | 1947 | 52 | M | Weight loss, indigestion | 7 years | None | Pyloric obst., old gastro-enterostomy | 60 Free 78 Total | Gastric resection | Edema, prolapse | Well 13 years |
| Appleby | 1947 | 44 | F | Epigastric pain, vomiting, weight loss | Years | None | Prolapse mucosa | 40 Free 70 Total | Gastric resection | Edema, prolapse | No follow-up |
| Appleby | 1947 | 57 | F | Cramping pain, fullness, wt. loss | 8 years | None | Prolapse mucosa | 2 Free 33 Total | Gastric resection | Gastritis, prolapse | Well 5 years later |
| Appleby | 1947 | 65 | F | Fullness, pain, weight loss, indigestion | 3 years | None | Prolapse, retention | 0 Free 60 Total | Gastric resection | Prolapse | Well 3 years later |
| Appleby | 1947 | 43 | M | Fullness, weight loss, epigastric distress | Years | None | Prolapse | 14 Free 65 Total | Gastric resection | Prolapse, mucosa | Objectively normal 10 months |
| Judd and Moe | 1947 | 53 | M | Fullness, indigestion | 18 months | None | Obstructing lesion | 0 Free 6 Total | Gastric resection | Prolapse, gastritis | Well at discharge |
| Nygaard and Levitan | 1948 | 45 | M | Indigestion | 13 years | Hematemesis | Prolapse | 0 Free 15 Total | Gastric resection | Chronic gastritis, healed erosion, gastritis | Well 1 month later |
| DiLeo, Kuker, and Shepard | 1948 | 65 | F | Epigastric pain, indigestion | 2 years | Hematemesis, melena | Prolapse, duodenal diverticulum | Not stated | Excision mucosa | Prolapse, chronic gastritis | Well 6 months later |

case,—that of hematemesis with secondary anemia,—seems to be an important finding.

CASE REPORT

M. R., 65 year old colored female, was admitted to Charity Hospital on December 1, 1947, with the chief complaint of having vomited about 500 cc. of coffee ground material the day before admission and later of passing several tarry stools. Further questioning revealed that she had intermittent attacks of epigastric pain, indigestion, and occasional attacks of nausea and vomiting for the past two years. Total weight loss 40 lbs.; however, her local physician had placed her on a low caloric diet for hypertension.

Physical examination revealed blood pressure of 160/80 mm. Hg. The mucous membranes were pale. An aortic systolic murmur was noted. Examination of the abdomen was essentially normal. Rectal examination revealed black stool on the examining finger, which gave a 4 plus benzidine test for blood. Pelvic examination negative. Multiple pigmented nevi scattered over body.

Laboratory studies: Hemoglobin 6.5 Grams; R.B.C. 2.5 million; W.B.C. 3,555; Polys. 60, Monocytes 6, Lymphocytes 34; Hematocrit 19. Urinalysis normal. Blood chemistry studies normal. Kline and Kolmer negative.

G. I. Series: (December 1, 1947.) No definite abnormalities demonstrated under fluoroscopy. The films demonstrate prolapse of gastric mucosa into the duodenum. (Fig. I) A diverticulum is noted in the region of the junction of the second and third portion of the duodenum. No gastric retention.

The patient was placed on a high caloric, high protein diet supplemented with vitamins, ferrous sulfate, and given a total of 1500 cc. of whole blood during the preoperative period, raising the hematocrit to 38 mm. On December 23, 1947, the patient was taken to surgery and under ethylene-oxygen-ether general anesthesia an exploratory procedure was performed through an upper right rectus muscle splitting incision. The liver, spleen, cecal, and pelvic regions presented no abnormalities. The stomach appeared and felt normal except for a boggy mass in the pyloric region, extending into the first portion of the duodenum. No other masses were palpable nor was the pyloric region fixed by any infiltrating growth. The stomach was brought into the field and opened by a 2" longitudinal incision in the prepyloric region. An Allis forceps was introduced through the pylorus and a mass of redundant prolapsed mucosa brought into view. This was resected in a circumferential manner and sutured, using an atraumatic chromic 00 continuous lock stitch suture, including an occasional bite into the deeper layers. The gastrotomy opening was then closed in a transverse manner with a double row of continuous chromic 00 atraumatic catgut and a seroserous layer of interrupted sutures of quilting cotton. No attempt was made to remove the diverticulum in the duodenum. The abdomen was closed in anatomical layers, using catgut technic.

Figure 1. Preoperative gastrointestinal series showing prolapsed gastric mucosa into the duodenal cap (indicated by arrow) presenting a mushroom appearance with a central streak as described by Eliason, Pendergass, and Wright.

The pathologist's report of the specimen: Tissue received in formalin measuring 7 x 3 cm. and 0.3 to 0.1 cm. thick. Mucosal surface wrinkled, smooth, and yellow. Serosal surface ragged and red. Microscopic diagnosis chronic gastritis.

Postoperative course was uneventful and the patient was discharged on the fourteenth postoperative day. A gastrointestinal series done on January 13, 1948, revealed no abnormality of the esophagus, stomach, or duodenum. (Fig. 2) The diverticulum is again noted.

Postoperative follow-up at four months: Patient had felt well since surgery, had been completely relieved of epigastric symptoms, and was eating a regular diet without difficulty. At six months follow-up she had gained 15 pounds in weight and was completely asymptomatic. A repeat gastrointestinal series again revealed the stomach and duodenum to be normal except for the diverticulum previously reported.

It is our impression that since the patient has remained entirely asymptomatic following surgery,

Figure 2. Postoperative gastrointestinal series reported by radiologists as normal filling of stomach, pylorus, and duodenal cap. No evidence of previous defect.

the diverticulum reported in the third portion of the duodenum was probably not the cause of her complaint.

### ETIOLOGY

The etiology of this condition is unknown. However, several theories have been offered.[2, 4, 6] The most attractive explanation to us is that there is first developed a hypertrophic gastritis with large redundant rugae in the prepyloric portion. These are gradually forced toward the pylorus by peristalsis, with resultant increased mobility of the submucous layer, until finally a fold is pushed through the pylorus into the duodenum. Symptoms are due to the partial block thus produced and the trauma to the mucosa causes superficial erosions which may bleed.

### SYMPTOMS

The symptoms usually noted are epigastric pain (56 per cent) and indigestion (47.2 per cent). This has been described as being increased rather than diminished by ingestion of food.[3, 8] Gastrointestinal bleeding as noted by hematemesis, melena, or persistent occult blood in the stools was found in 38.2 per cent. We consider this finding significant, and feel that every case of unexplained gastrointestinal bleeding should be investigated from this standpoint.[8] Symptoms of less constant occurrence are weight loss (17.2 per cent), vomiting (14.7 per cent), epigastric fullness (14.7 per cent), and weakness (6 per cent). Weight loss has been particularly emphasized by Appleby[6] and Norgore and Schuler.[3] As is apparent, these symptoms are nonspecific and may be imitated by a variety of upper abdominal diseases. Diagnosis, therefore, depends on roentgenological examination. In the collected cases a definite mucosal prolapse on x-ray was observed in 22 cases (64.7 per cent), and in all but one some type of prepyloric or bulb deformity was noted. It should be emphasized that retention is not necessary for the roentgenological diagnosis[9] and that simple prolapse cannot with certainty be differentiated from prolapsing tumors and malignancy.[1, 10] The symptoms are chronic and are apparently allowed to persist for some time before the patient seeks medical advice. In the reported cases 20 out of 27 waited more than one year from onset of symptoms before presenting themselves to their physicians.

TABLE II

| SYMPTOM | NUMBER | PER CENT |
|---|---|---|
| Epigastric pain | 19 | 56.0 |
| Indigestion | 16 | 47.2 |
| Gastrointestinal bleeding | 13 | 38.2 |
| Weight loss | 6 | 17.2 |
| Vomiting | 5 | 14.7 |
| Epigastric fullness | 5 | 14.7 |
| Weakness | 2 | 6.0 |

TABLE III

DURATION OF SYMPTOMS

| | |
|---|---|
| Less than 1 year | 7 cases |
| Over 1 year | 20 cases |
| Average duration | 51 months |

### PHYSICAL AND LABORATORY FINDINGS

The physical findings are not usually noteworthy. Localized tenderness is sometimes observed and there may be evidence of recent weight loss or anemia. Prolapse seems to be found more commonly in the

TABLE IV
AGE INCIDENCE

| Decade | No. Cases |
|---|---|
| 20-30 | 6 |
| 30-40 | 5 |
| 40-50 | 9 |
| 50-60 | 8 |
| Average Age | 45.8 years |

older age groups, the majority of cases occurring in the fifth and sixth decades. (Table IV.) The average age in this group was 45.8 year. Laboratory findings may demonstrate a mild or moderately severe anemia. The gastric analysis usually shows a normal acidity. (Table V.) There is a

TABLE V
ACIDITY

| | |
|---|---|
| Normal | 13 cases |
| Hyperacidity | 6 cases |
| Hypoacidity | 3 cases |
| Not indicated | 12 cases |

slight predilection for the male sex, there being 22 males and 12 females in the reported cases.

DIAGNOSIS

We believe that patients who have suffered abdominal distress in whom prolapsed gastric mucosa can be demonstrated and who fail to respond to a brief trial of medical management should have an exploratory laparotomy. This view is supported by several facts to which Bockus[17] calls attention by saying, "When complete prolapse into the duodenum occurs, the differentiation between pedunculated tumor and prolapsing gastric mucosa will often be impossible and operation may be necessary. If there is a large defect in the pyloric canal and a negative shadow in the duodenal bulb, the condition may not be distinguishable from that of pyloric ulcer, hypertrophic gastritis, or carcinoma." This difficulty in diagnosis [4, 10, 14] has been clearly demonstrated in two case reports, one by Melamed[4] in 1933, which presented a large ulcer in the prolapsed mucosa, and another report by Rubin,[91] in which the resected specimen revealed malignant change. Further evidence in favor of surgical management is the excellent results reported by Scott[11] on 5 cases with complete relief of symptoms and no recurrence on a full diet.

The types of operation (Table VI and Table I) used for this condition range from simple resection of the mucosa to gastroenterostomy and in some cases gastric resection. It is our opinion that the various procedures used all have certain merits which cannot be challenged, and that time and an increasing number of reported cases alone will permit a more adequate evaluation.

TABLE VI

| Procedure | Cases | Good Results | Poor Results | Not Followed |
|---|---|---|---|---|
| Excision of mucosa and pyloroplasty | 7 | 4 | 1 | 2 |
| Gastric resection | 10 | 8 | 1 | 1 |
| Excision of redundant mucosa | 6 | 6 | .. | .. |
| Gastrotomy | 1 | .. | .. | 1 |
| Excision of mucosa, gastroenterostomy | 3 | 1 | .. | 2 |
| Pyloroplasty | 5 | 2 | 3 | .. |
| Closure pylorus, gastroenterostomy | 1 | .. | 1 | .. |
| Section of pylorus, excision mucosa | 1 | 1 | .. | .. |

It is our opinion that abnormalities of this type should be reported by the roentgenologist, letting the further treatment rest with the clinician. A fair trial of medical management is indicated in all but the obstructed bleeding or suspected malignancy cases. Next it seems fitting and proper to perform the simplest surgical procedure that will give good results. Here we must depend on the judgment of the individual surgeon. If at the time of exploration gastric mucosal prolapse alone is found, simple excision and suture will suffice. If the mucosa is ulcerated or presents a tumor, a frozen section should be followed immediately by a gastric resection if malignancy exists.

SUMMARY AND CONCLUSIONS

1. Thirty-four cases of prolapsed gastric mucosa have been collected from the literature and reviewed, including one not previously reported. Only cases without tumor formation and proved at operation were used.

2. It is believed that this condition is more common than is reported and that it may be a cause of unexplained gastroin-

testinal bleeding. The symptoms are not diagnostic and the diagnosis must be made by x-ray. Retention is not necessary for roentgenological diagnosis.

3. Operation should be considered in patients with prolapsed gastric mucosa, who have upper abdominal complaints or gastrointestinal bleeding and who fail to respond promptly on brief medical therapy. Good results may be expected from surgery.

4. After gastrotomy is done to determine the nature of the lesion (and this should be done even when the pylorus is normal to palpation), the treatment of choice is either simple excision of the mucosa with or without pyloroplasty, or gastric resection.

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# THE TREATMENT OF HEAVY METAL POISONING WITH BRITISH ANTILEWISITE (BAL)*

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NEW ORLEANS

## HISTORICAL

No medical research developed during World War II has opened more fields of possible study than has that on the compound 2, 3-dimercaptopropanol (British antilewisite, BAL). It was developed by Peters and his associates[1] during the war years as an antidote to the arsenical blister gases. Its original use was a local one and intended to counteract superficial contamination of the cutaneous areas and of the mucous membranes of the eyes. Subsequent research found it to be most effective in combating systemic sequelae of arsenic poisoning, from exposure to both arsenical gases and ingested and injected arsenicals, taken therapeutically or as a poison. It has since been used as a prophylactic agent to prevent complications, or arsenic poisoning, when arsenicals are used medicinally as in syphilotherapy. The results in large series of the last type of cases indicate that many of the serious complications of arsenotherapy may be greatly reduced by the use of BAL when administered early and in proper amounts, intramuscularly.

BAL has since been used in cases of acute poisoning from various other metals. The results in acute mercury poisoning, as well as in gold poisoning, have been most encouraging. The studies of BAL begun at Oxford by Stockton, Thompson, Peters[1] and their associates have been carried on and developed in the United States since 1941.

## EXPERIMENTAL

The study of the action of BAL has resulted in an important advance in the understanding of fundamental biologic mechanisms. BAL is unstable to heat and acids and decomposes during distillation.

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The toxicity of trivalent arsenicals is largely due to their binding of essential thiol groups in enzyme proteins. This concept was supported by the significant observation that dithiols of the BAL type not only prevented the inhibition of enzyme systems by arsenicals but also reactivated these enzyme systems when added after inhibition had occurred. From the extensive research of various investigators,[2] here and abroad, on the effect of trivalent arsenicals, chiefly lewisite, on enzyme reactions concerned with cellular metabolism, it is concluded that trivalent arsenicals exert their toxic action by combinations with S. H. groups of the activating protein of enzyme systems. Tissue respiration is interfered with by the action of the arsenicals on the large groups of S. H. enzymes essential for carbohydrate transformation and fat metabolism. The enzyme inhibitions produced by lewisite and arsenic as well as certain other metals, can generally be prevented by BAL or other closely related dithiols.

BAL is a strong reducing agent and is rapidly oxidized in the presence of catalytic amounts of copper and hemin. BAL destroys the physiologic activity of insulin. These reactions of BAL may be associated with the toxicity of the compound when injected in large amounts.

### TOXICITY

BAL is not an innocuous substance; Peters and his associates[1] observed profound toxic effects. The striking physiologic and metabolic changes of BAL poisoning are not accompanied by comparable morphologic alterations. Little is to be seen grossly or microscopically, other than congestion of the viscera and occasionally, accumulation of fluid in the serous cavities or lungs[3, 4]. Lesions of the central nervous system have been described following the injection of certain BAL derivatives.[3] In man, solutions or ointments containing 5 to 10 per cent BAL produce only temporary lacrimation, blepharospasm and eye pain, when instilled into the conjunctival sac[4, 5]. Injected intramuscularly in sterile peanut oil and benzyl benzoate solution, concentrations of BAL of 10 per cent or less are well tolerated.[6-9] Inunction of a total of 1 cc. of undiluted BAL or of 2 gm. in a jelly base does not produce systemic effects in man.[8-10]

### CLINICAL USAGE

Working with the S. H. containing enzyme succinoxidase, it has been shown that heavy metals such as lead, antimony, bismuth, cadmium, mercury, and zinc produce complete inhibition and that the enzyme is reactivated on the addition of certain BAL derivatives. This demonstrates that the toxicity of these metals is like that of arsenic, due to the inhibition of the S. H. group. These studies indicate that there is need for experimentation designed to test the therapeutic value of BAL in these heavy metal intoxications. Subsequent clinical studies have added to the knowledge of drug intoxications which are greatly helped by BAL therapy; among these are gold and tellurium.

The most extensive investigation of the clinical value of BAL has been in arsenic poisoning and its complications. The largest series in this group has been reported by Eagle and co-workers and a summary of the findings in his 200 cases is included herein.

TABLE I

SUMMARY OF 200 CASES OF ARSENICAL POISONING TREATED WITH BAL (Eagle)

1. *Arsenical dermatitis*: In 88 cases, 80 per cent responded to treatment.
2. *Toxic encephalitis*: In 55 patients, 44 recovered completely within one to seven days and 11 died.
3. *Agranulocytosis*: In 11 cases, 10 recovered with 1 death.
4. *Aplastic anemia*: In 3 cases, there was no beneficial effect from BAL.
5. *Jaundice*: In 14 patients, 5 showed prompt response, 7 evidenced no effect, and in 2 the effects were debatable.
6. *Massive overdosage of mapharsen*: In 4 cases there were 3 with prompt improvement and 1 death.
7. *Arsenical fever*: There were 44 cases with prompt recovery in all.

### REPORT OF CASES

A group of 18 cases of arsenical intoxication in adults seen at Charity Hospital of Louisiana at New Orleans has been summarized and is reported. Serious compli-

TABLE II

ARSENICAL POISONING IN ADULTS TREATED WITH BAL

| Diagnosis | Age of Patient | Race | Sex | Total Dosage | Immediate Reactions and Comment | Complications |
|---|---|---|---|---|---|---|
| **Arsenic Poisoning (Ingestion)** | 19 | W | Female | 16.2 cc. | Response good | Disappearance of severe nausea after first Rx. |
| **Arsenic Poisoning (Ingestion)** | 42 | W | Male | 27 cc. | After 1st dose of BAL complained of: 1. Local pain. 2. Generalized weakness. 3. Headache-nausea. 4. Profuse sweating. 5. Burning sensation mouth and throat. | Symptoms began 3-5 minutes following ingestion. |
| **Arsenic Poisoning** | 16 | W | Female | 16 cc. | None—good. | None. |
| **Arsenic Poisoning (ant poison)** | 18 | C | Female | 2 cc. | No reactions. | None. |
| **Arsenic Poisoning (sodium arsenite mistake)** | 45 | W | Male | 12 cc. | No reactions. Response good. | None. |
| **Arsenic Poisoning acute** | 24 | C | Female | 24.5 cc. | No reactions. Response good. | None. |
| **Arsenic Poisoning subclinical** | 26 | W | Male | 6.6 cc. | None—good. | None. |
| **Acute Arsenic Poisoning (ant poison)** | 67 | W | Female | 45 cc. | No immediate reaction. Response good. | None. |
| **Arsenical Dermatitis** | 60 | C | Male | 20 cc. | Fever end of fifth day. | Fever-abscess. |
| **Arsenical Dermatitis** | 41 | C | Female | 34 cc. | No untoward reaction. Good results. | None. |
| **Arsenical Dermatitis** | 38 | C | Male | 34 cc. | Remarkable improvement on BAL. | None. |
| **Arsenical Dermatitis** | 43 | C | Male | 39 cc. | Good results. | Complained of headache during BAL administration. |
| **Arsenical Dermatitis** | 36 | C | Male | 164 cc. | Good response. | None. |
| **Arsenical Dermatitis** | 43 | C | Female | 38 cc. | Almost immediate loss of periorbital edema; rash rapidly subsided. | None. |
| **Arsenical Dermatitis** | 62 | W | Male | 30 cc. | Good response. | None. |
| **Arsenical Dermitiitis** | 46 | C | Male | 40 cc. | Good response. | None. |
| **Arsenical Sensitivity** | 28 | C | Female | 18 cc. | Patient ran fever 105° F. after arsenical therapy. | Temperature gradually receded to normal after BAL injections. |
| **Acute Arsenic Poisoning (ingestion)** | 19 | W | Female | 12 cc. | Patient took ant poison, had severe vomiting, diarrhea and albuminuria. | Disappearance of symptoms by third day. |

cations were conspicuous by their absence. The rapid response in all these cases was striking.

Norman C. Woody and John Kometani[11] recently read a paper at the Annual Meeting of the American Academy of Pediatrics, Dallas, Texas, December 1947, on BAL in the treatment of arsenical ingestion in children. The same authors also published a paper on the subject in the New Orleans Medical and Surgical Journal. These excellent papers showed that BAL is an effective chemical antidote for arsenic poisoning. The side effects in their series were not serious. These authors made recommendations concerning the application of BAL in the treatment of one of the most common and serious pediatric emergencies.

The prophylactic use of BAL in preventing reactions in intensive arsenotherapy when other antisyphilitic drugs cannot be used offers a new field of usefulness for this new drug.

Local applications of BAL ointment are of value either as a supplement to BAL injections, or in those patients in whom systemic BAL administration is contraindicated because of its severe side effects in such conditions as hypertension, and heart disease.

BAL properly administered is effective in the treatment of patients with arsenical encephalitis and in individuals who have received a massive overdose of mapharsen[12]. BAL is probably of value in some cases of *blood dyscrasia* resulting from arsenotherapy and it appears to be of little if any value in most cases of so-called arsenical jaundice[13].

### GOLD POISONING

Preliminary experiments on rats have shown that the compound formed by the union of BAL with gold is nontoxic. In 5 cases, twenty-four hour urine specimens showed definite increase in the excretion of gold after BAL treatment was begun. In 4 patients with eruptions of less than two months' duration there was prompt cessation of pruritis and the rash cleared within a month. The fifth patient who had had an extensive eruption for three months before BAL therapy did not respond to treatment. Presumably this patient was treated after the toxic changes produced by the gold had become irreversible, since the urinary excretion of the gold was equal to or greater than that found in patients showing satisfactory clinical response.

BAL is effective against both toxic and allergic manifestations due to gold. Toxic reactions to gold injections in doses used in rheumatoid arthritis are reported by Cohen, Goldman and Dubbs,[14] as occurring in 25 to 56 per cent of cases. I do not encounter many toxic reaction to gold injections in our skin clinics or in my private practice. This great incidence and variation in reactions encountered by many following gold injections is hard to understand. Perhaps the use of smaller doses, 5 to 50 mgms., once weekly, for a total dosage of about 765 mgm. would decrease the incidence of the gold reactions. Occasional sensitivity manifestations occur despite all precautions.

### POISONING FROM OTHER HEAVY METALS

In mercury poisoning, as was anticipated from preceding animal experiments, the results in treating these cases with BAL have been most encouraging[19]. The absence of clinical reports in bismuth poisoning is surprising since bismuth is in such general use today. The bizarre manifestations of bismuth intoxication and the difficulty in diagnosing it probably account for the absence of information on the use of BAL in bismuth poisoning. The use of BAL in cadmium and antimony poisoning has been favorably reported on. Further studies will probably add new usage for BAL as the drug is still a relatively new one.

### DOSAGE

Experiments showed the necessity for further studies in animals in regard to toxicity and therapeutic effectiveness of BAL in various vehicles, routes of administration, and dosage schedules.[16] The data so obtained made possible the development of a stable sterile preparation of BAL in benzyl benzoate-peanut oil solution, suitable for intramuscular injection. The most effective method of treatment was found to

consist of four injections of BAL at two to four hour intervals, followed by a single daily injection for six days. On this schedule, BAL in doses of 1 to 10 mgm. per kg. per injection saved 55 per cent of the animals from repeated massive doses of mapharsen and delayed death in an additional 22 per cent. The toxicity of this preparation on intramuscular injection in man was then carefully investigated. The result of these studies on dosage are as follows.

### DOSAGE OF BAL

The dosage of BAL is 2.5 mg. to 3 mg. per kilogram of body weight, 10 per cent in oil, to be given intramuscularly every four hours for the first few days, five injections on the third day, and two injections, daily, thereafter for ten days, or until recovery.

### REACTIONS AND COMPLICATIONS

It has been shown that BAL in oil injected intramuscularly, at a dosage level of 3 mgm. per kg. produces only the mildest reactions in a small percentage of the individuals tested.[6] If, however, the level is increased to 5 mgm. per kg. more than half the subjects experience some or all of the following reactions: nausea, vomiting, headaches, generalized aches and pains, burning sensations in the mouth, nose and eyes, sweating, restlessness, weakness, pain in limbs, jaws and trunk muscles. The heart rate is often increased, and there may be a rise in both systolic and diastolic blood pressure.[6-8] These signs and symptoms are transient and subside within four hours. Care should be exercised when BAL is given to patients suspected of impaired liver action. The pressure of severe renal disease does not appear to be a contraindication of BAL.

Prevention of reaction to BAL as given by Maurray Tye and John M. Seigel[17] was beneficial. Because of the close resemblance of severe symptoms caused by BAL therapy to those seen in patients with serum sensitivity, an injection of 0.6 cc. of a 1:1000 solution of epinephrine hydrochloride was given to a patient with arsenical dermatitis and hepatitis. Rapid and complete relief resulted when an injection of 25 mg. ephedrine sulphate was given orally before each ten subsequent injections of BAL, and no toxic symptoms occurred. A second patient was given 25 mg. ephedrine sulphate orally and had no toxic symptoms until the seventh injection when ephedrine was omitted. In a few minutes the patient noted severe lacrimation, headache, constriction of the chest, and a burning sensation of the side of the trunk. Subsequent injections of BAL were preceded by the administration of 50 mg. ephedrine and no further toxic symptoms were experienced. This is of eminent value when BAL therapy is imperative.

Kennedy and Henington[18] have reported a case in which the patient survived an accidental intravenous injection of an oil solution of BAL. BAL is a relatively safe drug. Most reactions are mild, and of short duration, and usually consist of profound weakness, nausea, vomiting, headache, muscular aches, lacrimation, and salivation.

### SUMMARY

Experimental and clinical results have been presented which prove that BAL is of definite value in the systemic treatment of various types of metal poisoning, particularly arsenic, gold, mercury, and antimony. The mortality and complications of poisoning from these metals are greatly reduced and recovery is greatly accelerated. The results obtained emphasize the importance of prompt and adequate treatment.

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# THE LOCAL INJECTION OF PENICILLIN IN ACUTE CIRCUMSCRIBED INFECTIONS

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Direct local injection of penicillin is an effective and economical method of administrating this therapeutic agent in acute localized infections caused by organisms sensitive to this antibiotic. To date, penicillin has proved to be the most effective agent against staphylococcic infections. It may be given intravenously, intramuscularly, applied topically or injected directly into an infected area: In 1948[10] we reported this method of treatment in over 300 cases of acute localized infections in various parts of the body. The results obtained were so highly satisfactory that it seems worth while to direct further attention to this method of treatment. The regional injection of penicillin in acute localized infections is not new: Florey[1], Cutler[2], Rose and Hurwitz[3], Peck[4], Sophian[5], Fisher[6], Kaplan and Rabin[10], and others, have employed this technic with excellent results. This work was initiated in 1943 on a septic surgical ward of an army regional hospital, and continued on the surgical out-patient services of Touro Infirmary and of Charity Hospital of Louisiana at New Orleans.

The purpose of this report is to record the observations and results obtained by the regional injection of penicillin in over 400 cases of acute localized infections in various parts of the body.

Acute infections may be divided for practical purposes into two clinical types: [7-10] (1) acute infections which do not cause death of tissue, such as septicemia and cellulitis; and (2) acute infections causing suppuration and death of tissue, as seen in boils and carbuncles. In the absence of suppuration or death of tissue, acute infections are superficial and spread over a wide area, relatively unconfined by a wall of inflammatory tissue. In contrast, where suppuration and death of tissue have occurred, a localized area of gangrene is surrounded by a zone of granulation tissue which separates the living from the dead tissue and which circumscribes the infection and prevents its spread, but which also acts as a barrier to free circulation into the diseased area. Because of these differences, the response to any form of therapy is not the same in these two types of acute infection.

It has been shown that the results of parenteral therapy with penicillin may vary considerably in the two types of acute infection. The results in acute infections in which there is no death of tissue are quite dramatic, while in acute localized infections, with suppuration, the results are not only less dramatic but at times even discouraging. If penicillin therapy is to be effective, the infecting organisms must be susceptible to the antibiotic, and adequate concentration of the drug must be obtained

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in the tissue in which the bacteria are imbedded. Because of the impenetrable inflammatory barrier surrounding a localized area of gangrene, circulation to this area is impaired and penicillin, when given parenterally, does not reach the diseased areas in sufficient concentration to be of any value.

Penicillin has several properties that give it distinct value as an antibacterial agent for local application. It is active against most of the organisms responsible for acute local infections. It is active in high dilutions. It is not inhibited by exudates, and it is nontoxic when given either locally or parenterally.

In view of these facts, it seemed to us that the most suitable method of administration of penicillin in acute localized infections should be direct injection into the infected area. By this means a high concentration and a deep, even distribution of the drug at the point where it was needed could be assured. The injections should be repeated often enough to ensure prolonged contact, thereby killing or inhibiting the growth of bacteria, so that the natural defenses of the body could complete the rest of the destruction and proceed with the process of repair.

### METHOD OF TREATMENT

Three cubic centimeters of distilled water or 1 per cent novacain are injected into the bottle containing 100,000 units of penicillin, making a dilution of approximately 33,000 units to the cubic centimeter. The size of the lesion determines the amount of the solution to be injected. Too much tension in the infected area may break down the barriers that the body has set up to localize the infection and must be avoided. This is a matter of judgment. Small lesions require 0.5 to 1 cc. at each injection, while larger ones, such as a carbuncle, may require 4 cc., or even more. The required amount is aspirated from the bottle containing the solution and the remainder kept in the ice box for the injection on the following day, as the potency of refrigerated penicillin in solution is maintained for approximately twenty-four hours.

The injection is made with a 24-gauge hypodermic needle into the most dependent area of the abscess. Without entirely removing the needle, the operator makes several injections into different areas of the lesion in order to insure an even distribution of the drug. Usually spontaneous evacuation of pus occurs during the injection, depending upon the stage of the infection. If the abscess is superficial and pointing, this occurs practically all the time. In cases in which the abscesses are fluctuant, they are aspirated with a larger needle immediately before the injection of the penicillin solution. In carbuncles the injections are made between the areas of pointing and the needle is usually inserted deeper than in the localized abscesses.

Following the injection a dry sterile dressing is applied. For several reasons it is desirable to keep the infected area closed. (1) This keeps the penicillin solution in contact with the infecting organisms. (2) It is important to obtain as high a concentration as possible in the diseased area, for in the early stages of infection there are some strains of staphylococci which are resistent to low concentrations of penicillin but are susceptible to the higher concentrations. (3) The danger of infection of the wound by secondary invaders which may produce penicillinase is minimized.

The injections are usually quite painful, the degree of pain varying with the density of the tissues and the volume of the fluid injected. However, the pain does not persist more than 4 to 6 hours.

Some increased redness is usually noted on the day following the injection. At that time another injection is made in the same manner as the first. On the third day the degree of resolution noted is quite surprising.

In about one-fifth of the furuncles only one injection was necessary; the others required two. Most were entirely healed on fourth day. The carbuncles required three, and occassionally four, injections, and were healed on the tenth day. In a large number of abscesses there was complete absorption without any external drainage.

RESULTS

*Carbuncles*: There were 60 carbuncles in this series; 21 on the posterior aspect of the neck, 28 on the upper back, 4 on the buttocks, 1 on the right anterior lateral abdominal wall, and 3 on the thighs, 1 on the chin, and 3 on the anterior chest wall. Three of these patients were severe diabetics. The average number of injections given on successive days was three. The average number of days required for healing was ten.

These results are in marked contrast to those observed following surgical procedures. An average of forty-three days was required for healing following incision and drainage in a series recently reported by Maes and Herringman[8, 9]. Extremely gratifying, in addition to the rapid healing time, was the cosmetic result obtained. There was practically no loss of skin and there were no deforming scars, as seen following surgical intervention.

A short control series was run in this group. Two patients with carbuncles on the neck were given 30,000 units of penicillin parenterally every three hours for seven days. At the end of this period both patients still had large carbuncles with many areas of drainage. It was six weeks following incision and drainage before healing took place. Two carbuncles on the back of the neck were injected locally with 100,000 units of penicillin in 3 cc. of distilled water daily for three days. In addition, 30,0000 units of penicillin were administered intramuscularly every three hours for ten days. The response was dramatic. The carbuncles seemed fairly to melt way and were entirely healed in ten days. Two carbuncles, one on the back of the neck, the other on the buttocks, were treated solely by local injection of 100,000 units of penicillin in 3 cc. of distilled water daily for three days. The results were equally as good as in the group which had been given penicillin parenterally as well as locally. In the two patients who received penicillin parenterally only, it is doubtful that the drug reached the infected areas in sufficient concentration to be of any definite value. Because of the inflammatory barriers surrounding the infection and the lack of circulation to the necrotic center, the antibiotic could not be expected to reach the bacteria imbedded in the slough and sterilize it. In contrast to the difficulty and expense of the maintenance of a high concentration of penicillin by parenteral administration over a period of many days or weeks, local injections offer a suitable and efficient alternative.

*Furuncles*: Furuncles formed the largest group in this series of cases. Eigthy-four per cent responded favorably to this form of therapy; 17 per cent required incision and drainage.

*Breast Abscesses*: Two abscesses of the breast were seen. They were both superficial, located near the nipple, and fluctuant. They were aspirated and injected with 60,000 units of penicillin in 2 cc. of distilled water on two successive days. Both were healed on the fifth day.

*Axillary Furunculosis*: There were 15 cases of axillary furunculosis. Ten patients had multiple lesions. All responded favorably and there were no recurrent "crops" as often seen following incision and drainage. The average time for healing, following two successive injections, was five days.

*Cellulitis*: While a fairly large number of cellulitides were seen, only a few were treated by local injections and this procedure was soon discontinued. These infections are superficial and not well localized. Any increased tension in them will cause the infection to spread. This happened in one of our patients when we first began this form of therapy. A young man was seen with an area of cellulitis measuring approximately 5 x 6 cm. on the ventral aspect of the right upper forearm. The area was injected locally with 60,000 units of penicillin in 2 cc. of distilled water. The following day the area of inflammation had spread to an alarming proportion. He was admitted to the hospital and, with the administration of penicillin intramuscularly, the lesion cleared in a short time. This type of infection responds rapidly to parenteral therapy and should be so treated.

### CONTRAINDICATIONS

Local injection of penicillin is not recommended in infections in the dangerous areas of the face, in cellulitides, and in bone felons.

### COMMENT

It is not the purpose of this report to leave the impression that this form of therapy should be used in all types of acute localized infections. It should be reserved for the more serious ones and for the lesions so located that surgical procedures may produce a bad cosmetic result. It is useful when surgical intervention has proved to be inadequate. In well localized infections that are fluctuant, simple aspiration or incision and drainage are the procedures of choice. Surgical judgment should be exercised in each individual case.

### SUMMARY AND CONCLUSION

1. The technic and the results of the local injection of penicillin in acute circumscribed areas of infection in over 400 ambulatory patients are presented.

2. Experience with this method of local therapy with penicillin has demonstrated that it is safe, effective, and economical. There were no complications.

3. Because of the impermeable inflammatory barrier surrounding an acute localized infection, penicillin, when given parenterally, may not reach a sufficient concentration in the affected area for therapeutic effectiveness.

4. By local injection it is possible to reach a concentration in the infected area far in excess of the highest levels obtained by intramuscular administration.

5. Following injection directly into the affected tissue, there is almost immediate improvement and recovery is established in a few days.

6. Extremely gratifying, in addition to the rapid healing time, is the cosmetic result obtained. There is practically no loss of skin and no deforming scars are seen as are usually found following surgical intervention.

7. This procedure is not recommended for the treatment of the cellulitides, for infections in the dangerous areas of the face, or for bone felons.

8. Surgical judgment must be exercised in each individual case.

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## THE MANAGEMENT OF PREMATURE LABOR

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If any single disease accounted for half of all adult deaths, it would receive extended scientific study and enormous lay as well as professional publicity. Yet premature labor which terminates between 5 and 10 per cent of all pregnancies and is associated with about half of all neonatal deaths receives little more than casual attention. Certainly no one can say that these infants are of negligible value in our civilization, for history has left us records of premature babies who have made their famous marks on the world[1]. How many more with names as celebrated as Charles

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Darwin, Victor Hugo, Voltaire and Winston Churchill might there be if more prematures had lived.

The most logical solution to the problem of the premature labor is to avoid it. For this very good reason many studies of the causes of premature labor have appeared[2-8]. (A review of the literature up to 1939 was published by Anderson and Lyon[9]). Toxemia, maternal uterine bleeding, multiple pregnancy and so-called "normal" premature labor are the common causes. Syphilis as well as other acute and chronic infections are of lesser importance. The gravest danger comes when several etiologic factors are active at the same time, e.g., bleeding plus toxemia plus syphilis. But we must be realistic and admit that little is known about prevention of bleeding and toxemia of pregnancy. Until these diseases are prevented, the incidence of prematurity will inevitably remain high. Tyson[2] has recently called attention to the fact that the incidence of premature birth in the Philadelphia Lying-In Hospital has remained essentially unchanged for fifteen years at an average of 9.4 per cent. This does not imply that premature labor cannot be prevented, for such important measures as careful prenatal care and adequate nutritution will diminish prematurity [10-12]. Nevertheless, at the present state of our obstetric development at least half of all premature labor is inevitable.

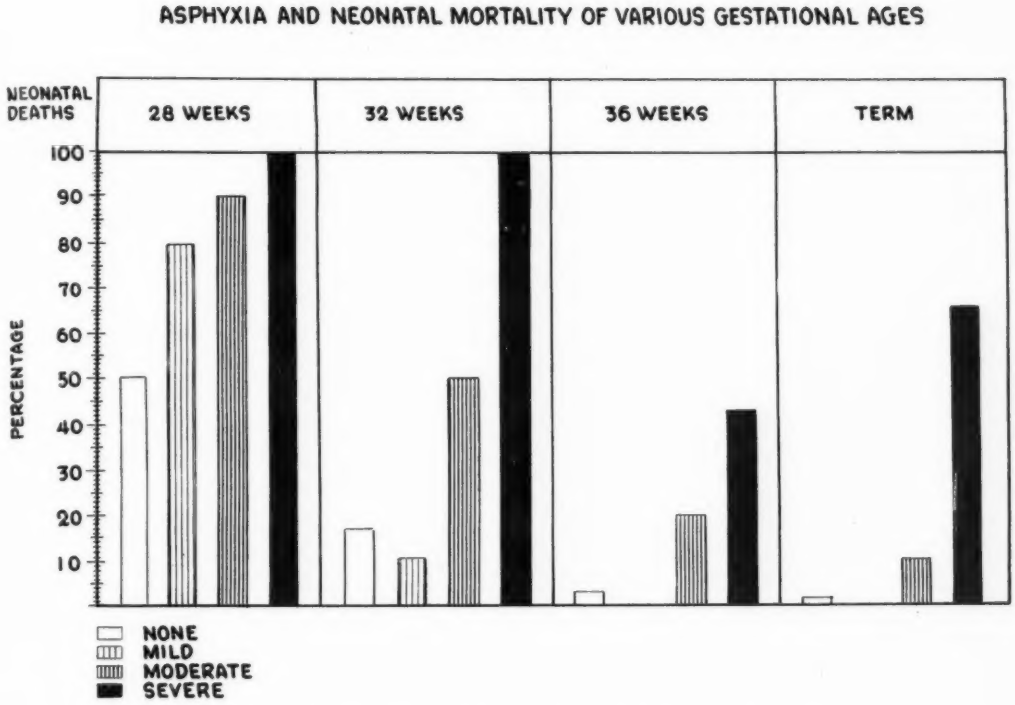
Although there has been a distinct decrease in the mortality of premature babies in recent years, a glance at vital statistics will show that most of the improvement has occurred in those babies who lived more than forty-eight hours. In general it is correct to say that deaths within the first forty-eight hours of life are of obstetric origin, while those after forty-eight hours are of pediatric origin. This does not necessarily imply culpability of the obstetrician. There is an inevitable and irreducible mortality of early life because of congenital maldevelopments and unpreventable immaturity. Nevertheless it is clear that many of the deaths without apparent cause, incorrectly ascribed to prematurity, can be prevented by proper obstetric management.

In many respects the anatomic and physiologic differences between the premature and mature infant are as great as those between the newly born mature infant and the adult. Herein lies some of our failure. Although the premature infant has many anatomic and physiologic handicaps,[13] a thorough understanding of four of these is essential in obstetrics.

### I. RESPIRATORY HANDICAPS

Several systems are at fault in this respiratory derangement. In the first place there is an anatomic immaturity. The alveoli of the lung are lined by a layer of cuboidal epithelial cells which separate the air of the alveolar space from the capillary endothelium. (The recent work of Joessyln indicates that in the mature individual the capillaries lie uncovered in the alveolar space.) Capillaries are infrequently seen. Furthermore there is little elastic tissue in the framework of the lungs as compared with the mature infant.[15] Physiologic immaturity is shown by the incompetent nervous control of respiration. Levine[14] exposed normal full term infants to an atmosphere containing 1.5 per cent carbon dioxide which caused a pronounced stimulation of respiration; premature infants similarly exposed were unaffected. Not until the concentration of carbon dioxide exceeded 4 per cent was stimulation of respiration observed. The paucity of capillaries in the brain may be responsible for the low reactivity of the respiratory center. The cough and gag reflexes are extremely weak, and the infant often makes little or no effort to overcome respiratory obstructions. Finally, the weakness of the muscles and bones of the thoracic cage combined with the lack of elastic fibers of the lung favors the maintenance of low intrathoracic negative pressure and atelectasis.

It is necessary for the obstetrician to manage properly this handicap. The part played by neonatal asphyxia is of major importance. There is an unbroken chain of events between pain relieving drugs and neonatal death. Premature infants tol-




erate analgesic agents very badly. Neonatal asphyxsia is more than doubled if the mother receives analgesics. Figure 1 shows the direct relation between the degree of neonatal asphyxia and neonatal deaths. Skillful use of pain-relieving drugs produces little serious asphyxia of the mature infant, but there is no place for these drugs in premature labor, however skillfully they may be given.[16] Many infants have been sacrificed because large or repeated doses of morphine or barbiturates were given in the vain hope of halting an inevitable premature labor. This, these drugs cannot do.

Local and regional anesthesia have much to offer in the proper conduct of premature labor. It is advisable to restrict the use of inhalation anesthesia at the time of delivery to small amount of the milder agents, such as nitrous oxide or ethylene, with plenty of oxygen.[17] Deep ether anesthesia is definitely contraindicated. Sometimes an argument against restriction of analgesics is raised by physicians who say that the patient demands it. Never have I found a mother who would not willingly accept such restrictions when the reasons were explained to her by the physician during the labor. If, in spite of preventive methods, the infant is asphyxiated at birth, proper treatment is essential. Above all, be gentle. All forms of violent resuscitation cannot be condemned too greatly; these include the Schultze swinging, jackknifing, cold tubbing, anal dilatation, dousing with ether, cuffing and slapping. Even holding the infant by the heels may be dangerous if anoxia is great, for the fragile capillaries of the brain are easily ruptured. If intubation of the trachea is necessary, it may be accomplished by passing a small tube under direct vision with the aid of a small laryngoscope or by tactile intubation. Most clinicians favor visual intubation, as the tactile method is not only difficult but also dangerous because of the small larynx and fragility of the mucosa and underlying structures.

Oxygen should be administered to every premature infant immediately after birth. It should be given whether or not there is visible cyanosis. The use of a carbon di-

oxide-oxygen mixture is advised by some men of note; many others favor the use of oxygen alone. To be efficient, the mixture would need to contain at least 5 per cent carbon dioxide, as premature infants are refractory to lower concentrations. Furthermore such a concentration of carbon dioxide would be too toxic for prolonged use, but it might be applied for short periods of time during the neonatal period to stimulate respirations.

II. TISSUE HANDICAPS

It is not necessary to discuss at length the obvious significance of immaturity and fragility of tissues. The state of the respiratory system just described is a part of this handicap. The brain, however, must have special mention. The gaping fontanelles and suture lines expose the brain to direct trauma, and intracranial hemorrhage is a serious complication in these infants.

The physician must diligently avoid trauma throughout labor and delivery. If induction of labor is necessary, it should be by medical means. Breese[18] found that surgical induction of labor was attended with a 42 per cent mortality as against 28 per cent for the medical method.

Once labor is established, oxytocics must never be used; sudden and explosive labors are dangerous for the infant. When for any reason the membranes remain unruptured, their integrity should be maintained as long as possible, even during the second stage of labor, as they cushion the head against the impact of pelvic soft parts.

There is an erroneous belief that cesarean section, being atraumatic to the fetus, offers the best chance for survival. Such is not the case. Clifford's[19] experiences, as well as our own, emphasize the poor results of cesarean section. The reasons for this are not entirely clear. The part played by general anesthesia in some of these deaths must not be overlooked. Breech delivery is equally dangerous. Beck[20] has called attention to the disproportionally large head of the premature infant, and suggests that the after-coming head is always delivered through a cervix incompletely dilated by the trunk and shoulders. The safest method is the vertex delivery from below with episiotomy when indicated. Some[19] have reported slightly better results with outlet forceps than with the spontaneous delivery. This might be a good plan in skilled hands especially when rigidity of the perineum in a primiparous patient causes delay in delivery; however, its value will be lost if a general anesthesia is a part of the procedure.

III. CIRCULATORY HANDICAPS

These are primarily hemorrhage and anemia. The fragility of the capillaries and the reduced prothrombin level may combine to produce excessive bleeding. Several factors produce capillary fragility; one common but often overlooked cause is anoxia. Anoxia in turn produces capillary hemorrhages in the lungs, adrenals, thymus and brain—hemorrhages which readily may prove fatal. A deficiency of elastic tissue about the small blood vessels makes them susceptible to minor degrees of trauma.

The anoxia may be qualitative, quantitative or both. Even though adequate numbers of erythrocytes are present at birth, many are of the nucleated type and are inefficient carriers of oxygen. Furthermore a serious anemia appears rapidly during the early neonatal period because of a deficiency of iron. These anemias are amenable to iron and to blood transfusions. In order to provide the maximum amount of hemoglobin and fluid to the premature infant, who will surely need them, it is advisable to delay clamping the umbilical cord until all pulsations have ceased. Further attention to the anemia becomes a part of neonatal care.

IV. METABOLIC HANDICAPS

Two metabolic handcaps of major importance concern this discussion. The first is birth weight. No single factor exerts greater influence on mortality than does the birth weight. No other pound can ever be as important to the life of the premature infant as the fourth one. For example, an infant who weighs 3 pounds has but one chance in four of survival, while an infant of 4 pounds has three chances in four.

There is but one way to increase birth weight: to prolong gestation. The importance of proper prenatal care, diet, and prompt attention to complications of pregnancy have been discussed.

At times premature labor must be induced for reasons of the mother's health. Immediately the obstetrician faces a two-fold dilemma: Shall he induce labor at the risk of the child for the sake of the mother or postpone delivery for the sake of the child but at the risk of the mother? The second, and probably more difficult question, concerns the survival of the infant *in utero*. Shall he interrupt a pregnancy at the risk of prematurity to save the fetus from possible, if not probable, death? This problem is not uncommon in toxemias of pregnancy especially in the hypertensive type.

The solution is not easy. Careful abdominal palpation augmented by roentgen studies will usually give a valuable estimate of the infant's weight. Careful and conservative management of most complications will permit some extension of the time of gestation. A delay of two or three weeks may increase the weight of the baby as much as a pound. Of course such delay must not be unduly hazardous for the mother.

The second important point concerns the inability to control body temperature. This erratic temperature control comes from an inability to regulate heat production as well as heat loss. Levine[14] accounts for the low heat production by the lack of crying and muscularly activity. Heat loss is excessive because of the large surface area of the premature infant in relation to weight and the lack of insulation—the subcutaneous fat. Finally the incomplete development of the nervous system, including the hypothalmus, may also derange temperature control.

The obstetrician plays an important part in the problem of temperature control which begins at the moment of birth. If the infant becomes chilled, immediately a drop in temperature follows. Ylppo[21] as well as others showed that the mortality of 1500-2000 gm. infants during the first five days of life was 8.6 per cent when the initial temperature was above 91.4° F as against 24.3 per cent in those below 91.4° F initially.

As soon as the cord ceases pulsating, it should be cut. Immediately the infant should be placed in a portable preheated incubator. At this time, and not before, the cord should be tied, mucus removed from the respiratory passages, and resuscitation given if necessary. During these procedures the infant should be exposed as little as possible, and oxygen should be given continuously. The infant in the incubator can then be moved to the nursery where constant attention of trained personnel is necessary and vital. Unless the infant can be weighed safely and with dispatch, such procedures can well be postponed until forty-eight or more hours have elapsed. Knowledge of the infant's weight does not add to the chances of survival, and the procedure may be hazardous. In fact, no value is gained, but the curiosity of the relatives, the physician, and the statistician is satisfied.

SUMMARY

The most logical solution to the problem of premature labor is to avoid it. Yet many of the diseases, such as toxemia, uterine bleeding, and multiple pregnancy which produce premature labor are as yet unpreventable. Certain causes of premature labor, such as syphilis and poor nutrition can and must be eliminated by proper prenatal care. Even with the best of care the incidence of premature labor will be distressingly high. It is essential then that premature labor be managed properly, keeping in mind four important physiological handicaps of the premature infant.

*Respiratory handicaps.* The anatomical and physiological respiratory handicaps are great and a high incidence of asphyxia neonatorum results. Pain relieving agents of all types are contraindicated because of respiratory depression. Morphine should not be used in an attempt to halt established premature labor. If anesthesia is necessary, it should be local or regional. All

violent efforts of resuscitation are dangerous. Resuscitation, when needed, should be by careful intubation. Continuous administration of oxygen is most desirable.

*Tissue handicaps.* Brain injury is common because of the fragile skull. Therefore membranes should be kept intact as long as possible. Medical induction of labor is safer than surgical induction. Oxytocics must never be used once labor is established. Spontaneous or low forceps vertex delivery is safer than any other method of delivery including cesarean section.

*Circulatory handicaps.* A qualitative or quantitative anemia is common. Therefore the cord should not be clamped until pulsations cease.

*Metabolic handicaps.* Birth weight is probably the most important factor which determines the mortality rate. The most important pound in life is the fourth because survival increases from 25 per cent to 75 per cent. Often it is possible to delay induction of premature labor to allow an increase in weight. The obstetrician must not allow the baby to become chilled at the time of delivery. Usually it is possible to have a pre-warmed incubator in the delivery unit. The premature infant need not be weighed immediately as this adds another unnecessary hazard. Careful attention to these principles will reduce premature mortality by one half.

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DISCUSSION

Dr. Myron E. Wegman (New Orleans): The high mortality rate of erythroblastosis fetalis and the development of methods for estimating the amount of antibody in the mother's serum have led to the proposal that erythroblastosis in the infant might be prevented by premature delivery by Cesarean section. It should be pointed out, however, that, in general, the risk of prematurity is greater than that from erythroblastosis. When one adds to this the risks of a section it appears preferable to wait until one has reasonable assurance of a fair sized baby and then to induce delivery from below. I should like to ask Dr. King and Dr. Lund to comment on this.

One other point is in regard to the advantages of replacement transfusion. Diamond and his group believe that this procedure is the treatment of choice because when one is properly set up, it is simpler than repeated transfusions and because it appears to shorten the hospital stay. On the other hand, replacement transfusion does not appear to lower the overall mortality rate from the disease.

Dr. E. L. King (New Orleans): I think our experience will substantiate that view. We have done six or eight sections for erythroblastosis just with that idea of getting the baby into the world earlier and getting it out of the unfavorable environment of an increase in titer and our results have not been satisfactory. We did not save any large number of babies in that way. We have felt that it

is better or at least just as well to let the patient go on to term and be prepared for a replacement transfusion as soon as it arrives if the condition is sufficiently serious and if not sufficiently serious, transfuse in the ordinary way as many times as may be necessary. Another thing that makes us take this attitude is that it is very difficult to correlate the antibody titer with the condition of the baby at birth. We have had some babies that have given considerable worry whose mothers had no antibodies, or some for only two or three weeks before the babies were born and still the babies have given us some difficulty. In other cases there was a considerable titer of antibodies in the mother's blood, still the babies were all right and required very little care. We have had, of course, one or two where the baby died in the uterus before delivery. I doubt if this could have been prevented by Cesarean section. One of those was a hydrops type of baby which would have been lost and we did lose one hydrops baby by Cesarean section.

Dr. Lund (In conclusion): What do you think of episiotomy for a seven and eight pound baby and for a four pound baby?

The question of episiotomy has come up in the handling of premature babies. I do not think we need to talk about episiotomy in the larger baby; that is almost like asking a person about his religion. However, the importance of episiotomy in small babies should not be underestimated. We know, for example, that in the primiparous patient with a fairly rigid perineum a small premature baby pounding against such soft tissues may suffer serious damage, especially in the brain. Some people have gone so far as to say that all premature babies should be delivered not only with benefit of episiotomy but also with low forceps and they have the results to back them up. I think, however, that if a general anesthetic becomes a part of the procedure, then certainly any advantage is lost. the multiparous patient who delivers readily need not have an episiotomy.

Obviously, at the present time we have no specific method of preventing premature labor. I would like to call your attention to such nonspecific things as diet. For example, some of the recent work shows that mothers who have adequate diet throughout their gestation have far less premature labor than others. Careful attention and adequate care of mild pre-eclampsia will keep it as mild pre-eclampsia rather than allow severe pre-eclampsia or eclampsia to develop. So the problem is not merely a specific one of injecting hormones, which has been suggested. The use of large doses of estrogens and progesterone have not been satisfactory in our hands.

NEW ORLEANS
**Medical and Surgical Journal**

*Established 1844*

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## THE IMPACT OF STATE MEDICINE ON THE PATIENT

Our patients should know how State Medicine will affect them. The most penetrating influence the medical profession can exercise against State Medicine is what each physician tells his patient. What the patient needs to know is how it will affect his life as an individual. The fact that the doctor feels an antagonism to being regimented is a novel and interesting concept to the patient but arouses no aggressive attitude in the patient.

Three places where the problem bears on the patient should be rubbed until they shine.

First, State Medicine will not be free. The cost will be met by an additional tax of 2 per cent or more on income paid both by employer and employees. As the actual expense is much more than the official estimates, this will soon be a 5 or 6 per cent tax on each. The first six months in England cost approximately 60 per cent more than was provided in the plan. That portion paid by the employer must still come out of earnings, or the firm could not remain solvent. We may anticipate then a 10 per cent tax on earnings. Dependable figures show that at present 4½ per cent of the family income goes to pay for medical care. The result, therefore, will be that free medicine under these conditions, without regard to quality, will cost the average family with an income of $3600 a year about $360, while now it costs $180. In New Zealand, where political medicine was set up less than ten years ago, the expense today absorbs 40 per cent of all revenues collected by the government, and deficit financing has been resorted to in a desperate attempt to furnish the benefits promised. Forty per cent of the comparable revenues of the United States would exceed $15,000,000,000 a year!

The second place where this Utopian planning rubs the patient is explained by Lenin's statement that in any Socialistic state socialized medicine is the keystone of the Communistic arch. There are now 15,830,899 persons paid each month by the Federal Treasury. Compulsory insurance will require 1 clerk for each 100 insured, or 1,400,000 bureaucrats. That means 17,500,000 votes on the Federal payroll. The winning party in the November 1948 election needed only 24,000,000. In such a situation State Medicine under any name would be an easy preliminary to nationalization of banks, utilities, mines, and transportation.

The third and most important aspect to present to the patient is the fact that socialized medicine will produce stagnation in the system that has made American

medicine the best there is. Those physicians who reflect on the matter know that bureaucratic domination will affect the quality of care. Others outside of the profession have studied the matter statistically. Noteworthy among these is the Research Council for Economic Security, Chicago; the result of this study has been published under the title of "Health of Nations." It is seen here that the system of paying for medical care does not in itself appear to play a major role in the health of the nation. This is based on such health indices as infant mortality, male life expectancy at birth, and death rates from selected causes. Studies were made in the records of countries having compulsory sickness insurance, including France, England, and New Zealand. These were compared with countries using the private fee for services system—which were the United States, Canada, and Australia. The study indicates, as might have been expected, that advancement will come by developing and expanding medical facilities, eliminating economic and racial handicaps, and by paying more attention to living standards, nutrition, and other such factors.

When the politician intervenes, the doctor-patient relationship suffers. When the incentive to do good medicine is removed, service becomes perfunctory, cold, and unadjusted to the individual. When alertness, consideration, and initiative are subordinated to routine the delicate flower of medicine withers.

# ORGANIZATION SECTION

**The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.**

***An informed profession should be a wise one.***

## SPECIAL MEETING

The special meeting of the State Society, called by the President, Dr. M. D. Hargrove, and held in Alexandria on February 20, was attended by a large group of physicians, their wives and lay people interested in combatting compulsory health insurance legislation proposed by the present Congress. The meeting, addressed by Honorable Forest A. Harness, of Kokomo, Indiana, Dr. R. B. Robins, of Camden, Arkansas and Mr. John F. McCloskey, of New Orleans, was an interesting one and it is felt that all present were made aware of the necessity for a unified fight against this vicious legislation.

There was some discussion concerning participation or nonparticipation in a government controlled plan, should such come into existence; however, the thought was expressed that this is not a topic for decision at this time; that if the proposed legislation is defeated there will be no need for consideration in this regard.

Immediately following the general meeting a luncheon was given for all in attendance. This was arranged for by the Rapides Parish Medical Society and special recognition was given members of this society for their cooperation in handling this as well as other details of arrangement for meetings during the entire day.

The House of Delegates convened at three o'clock and this meeting was open only to members of the House, members of the State Society and specified guests. The present situation in Washington was reviewed and the House of Delegates, by motion made and unanimously carried, reaffirmed the already expressed opposition of the State Society to socialized governmental, political and compulsory health insurance.

A protest sent to the AMA concerning activities of that Association in regard to the fight against socialized medicine, signed by 148 physicians, 17 of whom are members of the Louisiana State Medical Society, and also reply to this protest by the Board of Trustees of the AMA were read after which the following motion was made and car-

ried: That the House of Delegates go on record as condemning and disapproving action of these fifteen doctors of this state in signing this protest and that a copy of this action be sent to each doctor who signed the protest; also that a vote of confidence be extended the AMA and that discussion in regard to this matter be carried in the Journal. It was brought out in the discussion that it is felt doctors who signed this protest are attempting to sabotage efforts of the American Medical Association and other medical groups to defeat bills which will offer socialized medicine to the citizens of the United States.

The Chairman of the Council on Medical Service and Public Relations, Dr. A. V. Friedrichs, gave a report of the activities of the Council in an effort to have legislation calling for federalized medicine defeated, outlining what has been done and what is planned for the future in this regard.

Dr. O. B. Owens, President of the Louisiana Physicians Service, reported that representatives of the LPS have met with representatives of the Louisiana Hospital Service Association and the Hospital Service Association of New Orleans and that combination of these three plans has been considered. After discussion of this subject by Dr. Owens the following motion was made and carried: That the House of Delegates reaffirm its confidence in the Board of the LPS and instruct it to continue consultation and planning for organization of a unified statewide plan for selling voluntary insurance for both Blue Shield and Blue Cross coverage with the direction to preserve the sponsorship and vested interest of the Louisiana State Medical Society in the merger plan to be ratified by the House of Delegates as early as practical.

Dr. Guy Jones gave a report of the Committee on Rural Medical Service of which he is chairman and the following recommendation contained in the report was approved: That the Louisiana State Medical Society form a permanent committee to work with communities, make surveys and recommendations to the people of a community as to how they can obtain, support and keep a doctor in their community.

Attention was called to the fact that the new Social Security Bill contains a paragraph concerning medical care for the indigent along socialized medicine lines and motion was made and carried that a letter be sent to the senators and representatives from Louisiana requesting that they investigate this matter and vote against the bill on this basis.

A question was asked as to whether it is the opinion of the House of Delegates that doctors should continue to lend support to the National Physicians Committee and it was agreed that the work this group is doing is very worthwhile and should be supported in addition to the support given the AMA educational campaign.

It is hoped that every member of the Louisiana State Medical Society is as interested in protecting the American form of medical practice as the members in attendance at this special meeting which is considered one of the outstanding meetings held by this organization.

## NATIONAL CONFERENCE ON MEDICAL SERVICE CHICAGO, FEBRUARY 6, 1949

This conference opened on Sunday morning, February 6, at the Palmer House in Chicago, with considerable sadness due to the untimely death of Mrs. George Lull, wife of Dr. George Lull, Secretary-Manager of the American Medical Association.

Dr. E. F. Sladek of Traverse City, Michigan, President of the National Conference on Medical Service, reviewed the accomplishments of this conference during the past twenty-two years, and paid high compliment to the work that his predecessors had done. This was followed by discussion of "Legalized Medical Research" by two able leaders. Dr. Christ J. D. Zarafonetis of the University of Michigan reviewed in detail the medical problems involved in medical research. He stressed the point that investigators should be properly trained, properly equipped and should not

be handicapped by financial problems or other matters that might hinder this work. He added that certain goals should be attempted in any sort of reseach work. Many research problems, he said, are carried out very well but are worthless and should probably have not been attempted in the first place. Dr. George Wakerlin of the University of Illinois, discussed certain legal problems in medical research. He stressed that antivivisectionists were obstructing medical research on many fronts. He stated that some states actually have laws prohibiting animal experimentation and gave certain definite qualifications that must be met before research is carried out on human beings.

The next subject, "Can Corporations Such as Hospitals Legally Engage' in the Practice of Medicine?", was ably discussed by Dr. Wilbur Bailey, Los Angeles, California. He stated unequivocally that since hospitals or corporations do not have licenses, they are not legally practicing medicine. He described in detail how some of these institutions have tried to circumvent the law. In most cases, he stated, these groups should be prosecuted.

"The Progress of the World Health Organization" and "The Progress of the World Medical Association" were discussed ably by Drs. Frank Calderone, Director, American Office, Washington, D. C., World Health Organization, and Creighton Barker, Executive Secretary, Connecticut State Medical Association, respectively. The fact that world medicine outside the western hemisphere is in such a dilapidated state makes it seem impossible that anyone could possibly want to go into socialized medicine. "The Medical Program of the United Mine Workers of America Welfare and Retirement Fund" was discussed at length by Dr. Warren F. Draper. He planned with the doctors of America to help make this plan work. I think it is necessary for us to realize that Mr. John L. Lewis has come to the doctors for good medical care. Dr. Draper pointed out that they contemplate no other program. Certainly, they do not want any compulsory health insurance.

At the noon program, Dr. Howard, assistant to Dr. George F. Lull, gave a report concerning the American Medical Association's public educational program. He stated that every woman in America should read "Uncle Sam, M. D.", a recent publication mailed to wives of the doctors.

At the afternoon session, Dr. James R. McVay, Kansas City, Missouri, Chairman, Council on Medical Service of the AMA, told how the Council is trying to streamline its services to meet problems that arise.

Mr. Clem Whitaker, Public Relations Counsel for the AMA Coordinating Committee, spoke on "Implementing the New AMA Program". He stressed the fact that any program that succeeds has to start at the "grass roots". When Mr. Oscar Ewing predicted that the AMA would place a high-powered lobby in Washington, he certainly "missed the boat", according to Mr. Whitaker. He stated that if we can get 140,000 doctors and their wives to work in spreading this message to the people of America, socialized medicine will be whipped. He stressed, again, the fact that socialized medicine is just the opening wedge for *general socialism*.

Dr. Joseph S. Lawrence, Director of the Washington Office of the AMA reported what is going on in Washington at this time and he stressed the fact that if we are going to do anything about socialization of medicine, we must do it within the next ninety days. This came directly from a senate leader, who is against socialized medicine.

A panel discussion on "Postgraduate Education of the Doctor", by Dr. George N. Aagaard, of the University of Minnesota; Dr. C. W. Smith of Harrisburg, Pennsylvania; and Dr. Harold I. Goldman, Denver, Colorado, was conducted. Dr. Smith stated his state society in cooperation with the University of Pennsylvania is carrying postgraduate eduaction to the rural physicians. Their program is similar to the one being carried out by Dr. S. S. Chipman of Louisiana, but is on a more extensive scale. Dr. Goldman's survey on "Specialization in

Colorado" was very enlightening. He lauded the general practitioner and emphasized the fact that we should encourage more men to enter this field and to give them the credit which they are justly due.

Dr. Sensenich, President of the AMA, reported that doctors will not be drafted at the present time. He stated this decision had been made just prior to this meeting by Mr. James F. Forrestal, Secretary of Defense.

The meeting, which was considered very worth while, adjourned after election of officers.

J. P. Sanders, M. D.

### AMA ASSESSMENT

We are very glad to report wonderful response by the members of the Louisiana State Medical Society in contributing to the $25 assessment fund of the AMA. This is an indication of a very fine spirit of our profession by coming to the aid in such an important endeavor. We would urge all of those who have not contributed to do so as promptly as possible because the defeat of federalized medicine is definitely predicated on the amount of education on a national scale which can be produced with your money through the AMA.

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## LOUISIANA STATE MEDICAL SOCIETY NEWS

### CALENDAR

### PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

| Society | Date | Place |
|---|---|---|
| East Baton Rouge | Second Wednesday of every month | Baton Rouge |
| Morehouse | Second Tuesday of every month | Bastrop |
| Natchitoches | Second Tuesday of every month | |
| Orleans | Second Monday of every month | New Orleans |
| Ouachita | First Thursday of every month | Monroe |
| Rapides | First Monday of every month | Alexandria |
| Sabine | First Wednesday of every month | |
| Second District | Third Thursday of every month | |
| Shreveport | First Tuesday of every month | Shreveport |
| Vernon | First Thursday of every month | |

### SECOND DISTRICT MEDICAL SOCIETY

The regular meeting of the Second District Medical Society was held at Jill's Restaurant in Kenner, Louisiana, February 21 at 8 P. M., Dr. Joel Gray, President, presiding. Dr. Guy R. Jones of Lockport, president of the Louisiana section of the American Academy of General Practice, spoke on the present threat of socialized medicine. He also discussed the aims and activities of the American Academy of General Practice.

A full report was given by members of the Society who were present at the special meeting of the Louisiana State Medical Society in Alexandria on February 20.

### THIRD DISTRICT MEDICAL SOCIETY

A meeting of the Third District Medical Society was held on February 10 in Lafayette. The Society elected its officers for 1949. Professor John F. McCloskey, Dean of Loyola School of Pharmacy, addressed the group on the dangers of our form of Government if it embarks on a program of compulsory health taxation. Eleven members of the clergy of different faiths were guests at the meeting. They participated in discussion and made recommendations as to how such legislation can be combatted.

The possibility of establishing a Founders' Day scientific meeting in New Iberia was considered. This would commemorate the founding of the Attakapas Medical Society in that city on November 10, 1846.

### AMERICAN ACADEMY OF GENERAL PRACTICE

The Orleans chapter of the American Academy of General Practice held its regular monthly meeting January 31, 1949 at the Tulane Hutchinson Building. The members were privileged to hear Dr. E. H. Lawson of Tulane University present an interesting talk on laboratory procedures to be followed in the course of therapy.

Dr. A. Whife's resignation as treasurer was accepted at this meeting and Dr. Eugene Claverie was elected by acclamation as the new treasurer.

A dinner meeting is planned in March in place of the regular February meeting.

## DIABETES ASSOCIATION OF LOUISIANA

Dr. A. A. Herold, temporary chairman of the Diabetes Association of Louisiana, has called a meeting of that group for Saturday, May 7, in New Orleans. Further information may be obtained by addressing Dr. A. A. Herold, 1130 Louisiana Avenue, Shreveport, La.

## DOCTOR NEEDED

There is need for a doctor in the town of Cameron, Louisiana. The owner of a retail drug store there is desirous of securing a capable physician for this locality and will build an office in which he may practice. There is a population of 1500 in this town. A grammar school is located in the town and a high school is in Creole, Louisiana, which is 13 miles from Cameron and easily accessible by a good highway. Churches in the town are Catholic, Baptist and Methodist. For further information communicate with Mr. Albert Colligan, Cameron Drug Store, Cameron, Louisiana.

## POSTGRADUATE COURSE IN PEDIATRICS

A postgraduate course in pediatrics, sponsored by the Louisiana State University School of Medicine, will be held between March 21 and 25 in Room 610 of the school.

## PAN-AMERICAN CONGRESS ON MENTAL DEFICIENCY

The seventy-third Annual Meeting and Pan-American Congress on Mental Deficiency will take place on April 26-30, at the Roosevelt Hotel in New Orleans. For further information address Lou Kennedy, Ph. D., Chairman, Louisiana State University, Baton Rouge, La.

## UROLOGICAL POST GRADUATE SEMINAR

The American Urological Association through its Southeastern Section announces a Urological Post Graduate Seminar to be held in New Orleans, Louisiana, April 18, 19, 20, and 21, 1949. The Seminar will be under the auspices of the Division of Graduate Medicine, Tulane University School of Medicine. William D. Frye, M. D., Dean of the Graduate School of Medicine will be the Director of these courses in collaboration with the officers and Executive Committee of the Southeastern Section and with the representative of the Central Committee.

The course is designed especially for young urologists, urological residents, surgical interns especially interested in urology, and physicians and surgeons who do diagnostic urology (part time). It will be of especial value to those preparing for the American Board of Urology, but will afford an excellent review for all urologists.

The Seminar includes four full days, Monday, April 18, through Thursday, April 21, and will cover Anatomy, Embryology, Pathology, Physiology, Biochemistry, Endocrinology and Bacteriology. The courses will be given by men who are experienced urological teachers. They will present their subjects in an illustrated and attractive manner. Operative Urological Clinics will be provided on the various services of the hospitals for those who wish to stay over Friday, April 22.

The courses will be limited to 150 registrants. The cost will be $50.00 except for urologic residents. Because of the great amount of interest already expressed by members in our Section, an early application is recommended.

Address inquiries and applications to:

Wm. W. Frye, M. D., Dean
Graduate School of Medicine
Tulane University
New Orleans, Louisiana

Dr. Hugh T. Beacham of New Orleans, Louisiana is your state's representative on the Executive Committee of the Post Graduate Seminar of the Southeastern Section of the American Urological Association and he will be glad to receive communications concerning the course.

## NEWS ITEM

Dr. George M. Haik addressed the ophthalmologists of Birmingham, Alabama, at the University of Alabama Medical School in January. The subject of his address was, "Treatment of Glaucoma."

## PROSPECTIVE BUREAUCRATIC OPPRESSION OF DOCTORS

In a sharply-worded statement, commenting on Washington news reports that the Federal Administration plans to launch anti-trust prosecutions against several State Medical Societies, concurrent with the opening of its drive for compulsory health insurance, the American Medical Association today announced that "We will take our case directly to the American people if we find the Government is engaged in political persecution instead of legitimate prosecution."

Dr. Elmer L. Henderson, Cairman of the Board of Trustees of the American Medical Association, declared:

"The American people will not take kindly to Gestapo activities and the doctors of this country, when the health of their patients and the welfare of their profession are at issue, will not be frightened into non-resistance by threats against them.

"We have been making our own inquiry into the activities of Government investigators and if we find that an attempt is being made to use the Justice Department for political purposes, in an effort to stifle opposition to the socialization of medicine, we will air the facts to the people and demand a Congressional investigation into such activities.

"It is both false and absurd to imply that there is a monopoly in health insurance under the hundreds of voluntary systems operating in America today, with more than 52,000,000 insured mem-

bers. But there certainly would be an iron-clad monopoly if Patent Medicine Man Oscar Ewing got through his compulsory health insurance scheme and took over control of medical practice in this country.

"The American Medical Association is vigorously supporting all sound voluntary health insurance systems and is encouraging competition, between the pre-paid medical and hospital plans and the private insurance indemnity companies, because we believe the American people will get better coverag, at a better price, if many competing plans are available. We don't believe the people want a Government monopoly in the health insurance field, any more than they would condone a private monopoly, and we intend to fight the Government monopoly, proposed under compulsory health insurance legislation, even if the Federal Administration resorts to terroristic practices and witch-hunting in an attempt to frighten off opposition."

## FELLOWSHIP OF THE AMERICAN COLLEGE OF CHEST PHYSICIANS

The Board of Examiners of the American College of Chest Physicians announces that the next oral and written examination for Fellowship will be held in Atlantic City, June 2, 1949. Candidates for Fellowship in the College, who would like to take the examinations, should contact the Executive Secretary, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

The Fifteenth Annual Meeting of the American College of Chest Physicians will be held at the Ambassador Hotel, Atlantic City, June 2-5, 1949. An interesting scientific program has been arranged for this meeting and speakers from several other countries are scheduled to appear.

## FOUNDATION PRIZE by SOUTH ATLANTIC ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS

The South Atlantic Association of Obstetricians and Gynecologists announces the establishment of "The Foundation Prize." Authors of papers on Obstetrical or Gynecological subjects desiring to compete for the prize may obtain information from Dr. E. D. Colvin, Secretary-Treasurer, 1259 Clifton Road, N. E., Atlanta, Ga.

## ARMY MEDICAL DEPARTMENT ANNOUNCES DEVELOPMENT OF "DRAMAMINE," SEASICKNESS PREVENTIVE AND CURE

Working in conjunction with civilian investigators, the Army Medical Department has sponsored development of a new drug, "Dramamine," that acts as both a cure and preventive of seasickness or motion sickness, it was announced by Major General Raymond W. Bliss, The Surgeon General.

Credit for the original research is given to Dr. Leslie N. Gay, of the Protein Clinic of Johns Hopkins University Hospital, Baltimore, Maryland, who first began research on the drug in 1947, and Dr. Paul Carliner, also of Johns Hopkins.

In experiments recently completed, almost total cure or prevention of seasickness, in all degrees of severity, was obtained among more than 400 passengers aboard an Army transport in heavy seas.

Both the preventive and curative values of the drug in relation to seasickness were investigated during the voyage. The physicians reported that of the men who received preventive treatment, less than two per cent became seasick. In the therapeutic tests, the drug failed to give complete relief in only three per cent of cases.

During the extremely rough voyage, a total of 418 cases, including relapses of moderate to violent seasickness, were treated with Dramamine. Complete relief was obtained in 407 cases, with partial relief or failure in 11 cases.

Careful observation was made for unpleasant symptoms, but in not one instance, even though thousands of capsules were administered to more than 300 men, was there a complaint or evidence of discomfort which necessitated discontinuance of treatment.

Seasickness has been an important military problem because of the frequent necessity of transporting great numbers of men by air or sea and landing them in excellent physical condition. Especial attention was paid to the problem during World War II, in the course of which many drugs were used in an attempt to control its symptoms.

The drug was used extensively during the summer of 1948 aboard the U. S. S. America. Sufficient data was collected to warrant more extensive and intensive study of the drug. A brief report on the study was submitted to the Chief of Staff and The Surgeon General of the Army.

The Army secured the services of the U. S. Army Transport Ballou, a ship built for service in the relatively calm waters of the South Pacific. In order to try the drug under conditions most likely to produce seasickness, the Ballou was commissioned to carry 1,376 troops from New York to Bremerhaven, Germany, in November of last year. The North Atlantic is extremely rough and stormy at this season, and the vessel, which has more pitch and roll than ships designed for the rough waters of the Atlantic, experienced lists up to 36 degrees, which would tend to cause seasickness among even the hardiest sailors.

Four adjacent sub-level compartments, in which 485 men were quartered, were chosen so that all subjects would be exposed to the same motion of the sea. The men were divided into two groups. One group was used in a study of the drug's preventive qualities, and the other was studied to determine the curative qualities.

The men chosen for the preventive study were divided into two groups. One of these received 100 mg. of Dramamine in capsule form as the transport left New York. A similar dose was given six hours later and then one before each meal and one before retiring. The other group received a capsule containing only sugar on exactly the same schedule. Only Dr. Gay and Dr. Carliner knew who received the drug and who the sugar.

This schedule was continued for 48 hours, and then the administration of capsules was discontinued.

Of the 134 men who received Dramamine, none devolped nausea or vomiting while taking the drug; ony two men complained of dizziness. The physicians reported that the men maintained excellent morale, even complaining that they were unable to get enough to eat.

Of the 123 men who received the sugar capsules, thirty-five became seasick within 12 hours at sea. When placed on the Dramamine schedule the men in this group, with only one exception, derived complete relief within three hours.

In the compartment where Dramamine had been given from the start but its administration discontinued after 48 hours, 41 men reported that seasickness had developed 10 to 18 hours after the drug was omitted. The drug again was given to these men and 40 regained their normal state of health within 30 minutes to one hour after the first dose.

The group selected for the therapeutic trial did not receive any of the drug at the start of the voyage. Fifteen men became seasick, and 12 of these were immediately relieved after administration of Dramamine.

A sub-group of 33 men received sugar capsules. Nineteen men whose complaints had been nausea and dizziness were relieved within 12 hours by the sugar capsules. They were taken off the sugar capsules and remained well. Fourteen men became progressively worse on the sugar capsules, and complained of excessive nausea, extreme dizziness, and prolonged vomiting. After Dramamine was given, complete relief followed within half an hour after the first dose.

Other men aboard the ship became ill, 195 reporting severe symptoms of seasickness. Of this group, 187 were completely relieved within an hour after administration of the first capsule.

A number of men were so ill they could not retain the capsule in the stomach. The drug was given by rectum and within an hour they were able to retain both fluids and solid food.

All previous remedies had been combinations of various drugs, such as scopolamine, one of the barbiturate preparations. Dramamine is a single chemical which is believed to have a direct effect on the vomiting center in the brain. It is a member of the chemical family of benadryl and pyribenzamine, which are used in the treatment of certain allergic conditions. The complete chemical name is beta-diaminoethyl benzohydryl ether 8-chlorotheophyllinate.

Future plans call for broadening of experiments with Dramamine to include such means of travel as landing craft, small boats, and aircraft.

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## NEW OFFICERS OF COMPONENT SOCIETIES

*Assumption Parish Medical Society*
President—Dr. Henry A. LeBlanc, Paincourtville
Vice-Pres.—Dr. H. C. Dansereau, Labadieville
Sec.-Treas.—Dr. Julius W. Daigle, Paincourtville

*Bossier Parish Medical Society*
President—Dr. D. C. McCuller, Bossier City
Vice-Pres.—Dr. E. Scott Coyle, Plain Dealing
Sec.-Treas.—Dr. John B. Hall, Benton

*East Baton Rouge Parish Medical Society*
President—Dr. Charles McVea, Baton Rouge
Vice-Pres.—Dr. Charles Mosely, Baton Rouge
Sec.-Treas.—Dr. Daniel J. Fourrier, Baton Rouge

*E. & W. Feliciana Bi-Parish Medical Society*
President—Dr. B. S. Nolan, Jackson
Vice-Pres.—Dr. Paul Jackson, Clinton
Sec.-Treas.—Dr. E. M. Toler, Clinton

*Franklin Parish Medical Society*
President—Dr. Johnny Bostick, Gilbert
Vice-Pres.—Dr. A. J. Reynolds, Winnsboro
Sec.-Treas.—Dr. H. E. Jones, Wisner

*Iberia Parish Medical Society*
President—Dr. I. W. Gajan, Jr., New Iberia
Vice-Pres.—Dr. D. E. Bourgeois, New Iberia
Sec.-Treas.—Dr. L. Slipakoff, New Iberia

*Lincoln-Jackson-Union Parish Medical Society*
President—Dr. T. A. Dekle, Jonesboro
Vice-Pres. (Lincoln)—Dr. H. S. Roan, Ruston
Vice-Pres. (Union)—Dr. C. C. Colvin, Bernice
Sec.-Treas.—Dr. W. H. Kimbell, Ruston

*Orleans Parish Medical Society*
President—Dr. J. Kelly Stone, New Orleans
Pres. elect—Dr. C. J. Brown, New Orleans
1st Vice-Pres.—Dr. Boni J. DeLaureal, New Orleans
2nd Vice-Pres.—Dr. Sam Hobson, New Orleans
3rd Vice-Pres.—Dr. John J. Irwin, New Orleans
Secretary—Dr. N. J. Tessitore, New Orleans
Treasurer—Dr. J. O. Weilbacher, Jr., New Orleans
Librarian—Dr. Eugene H. Countiss, New Orleans

*Ouachita Parish Medical Society*
President—Dr. A. S. Hamilton, Monroe
Vice-Pres.—Dr. George A. Varino, Monroe
Sec.-Treas.—Dr. James W. Schonlau, Monroe

*St. Martin Parish Medical Society*
President—Dr. S. D. Yongue, Breaux Bridge
Vice-Pres.—Dr. Bernard de Mahy, St. Martinville
Sec.-Treas.—Dr. Murphy Martin, St. Martinville

*Terrebonne Parish Medical Society*
President—Dr. Samuel C. Collins, Houma
Sec.-Treas.—Dr. Buford J. Autin, Houma

*Vernon Parish Medical Society*
President—Dr. Edwin H. Byrd, Leesville
Vice-Pres.—Dr. T. S. Whitecloud, Leesville
Sec.-Treas.—Dr. W. H. Broyles, Leesville

*Third District Medical Society*
President—Dr. I. W. Gajan, Jr., New Iberia
Vice-Pres.—Dr. H. C. Voorhies, Lafayette
Sec.-Treas.—Dr. Thomas Latiolais, Jr., Kaplan

*Fourth District Medical Society*
President—Dr. W. C. Gray, Springhill
Vice-Pres.—Dr. R. H. VanHorn, Mansfield
Sec.-Treas.—Dr. J. E. Holoubek, Shreveport

## WOMAN'S AUXILIARY TO THE LOUISIANA STATE MEDICAL SOCIETY

### WOMAN'S AUXILIAY

The Executive Board of the Woman's Auxiliary to the Louisiana State Medical Society met in Alexandria, February 19, 1949, at the Bentley Hotel. The meeting was conducted by Mrs. O. B. Owens, President. (Mrs. Arthur A. Herold, Shreveport, Treasurer of the Woman's Auxiliary to the AMA was an honored guest). Reports were given by the twenty-five members present and several recommendations were made by the Executive Committee and passed by the Board to be presented to the delegates at the Convention. The following members were elected to serve on the Nominating Committee to meet before the Convention: Mrs. Rhodes Spedale, Chairman, Plaquemine, Mrs. A. Dent Tisdale, Monroe, Mrs. Wiley A. Dial, Baton Rouge, Mrs. Clarence Erickson, Shreveport, Mrs. George Taquino, New Orleans; Alternates, Mrs. M. C. Wiginton, Hammond, Mrs. A. Scott Hamilton, Monroe, Mrs. Dorman B. Barber, Alexandria, Mrs. Felix Boizelle, Baton Rouge, and Mrs. Cassius Peacock, New Orleans.

Members of the Board attended a luncheon at the Bentley Hotel arranged by the Chairman, Mrs. L. D. Gremillion.

The Convention Chairman, Mrs. C. Grenes Cole, announced the following program of the Woman's Auxiliary to be held in conjunction with the Louisiana State Medical Society, May 5, 6, 7, 1949.

Thursday May 5, 1949

Registration
Pre-convention Board Meeting.

Friday May 6, 1949

General Session—Mrs. O. B. Owens presiding
Tea and Style Show at Orleans Club in honor of Mrs. John Dunn, incoming President.

Saturday May 7, 1949

Postconvention Board Meeting
Luncheon—New Orleans Country Club.

The Woman's Auxiliary and its members who attended the Special Meeting and luncheon February 20 are deeply grateful for the invitation from the Medical Society to hear the addresses given by Dr. R. B. Robins, member of the AMA Council, Hon. Forest A. Harness, member of the 80th Congress, and John F. McCloskey, Dean, Loyola University School of Pharmacy.

Haddon Hall will be the headquarters for the Annual Meeting of the Woman's Auxiliary to the American Medical Association, which will be held in Atlantic City, New Jersey, June 6 to 10, 1949.

Requests for reservations should be sent at once to Dr. Robert A. Bradley, Chairman, Subcommittee on Hotels, 16 Central Pier, Atlantic City, New Jersey.

Members of the Woman's Auxiliary to the Orleans Parish Medical Society were entertained at their February meeting by Mrs. Frances Griffin Bres with violin selections. Among those attending the program and reception were members of the Executive Board of the Dental Auxiliary to the Louisiana Dental Association. The president, Mrs. E. A. Socola, has announced that the March meeting will be a Fashion Show and Reception with the wives of doctors who attend the Graduate Assembly as guests.

A Forum on Cancer was the February program for the Woman's Auxiliary to the Shreveport Medical Society. The President, Mrs. Jos. E. Heard has announced a new Speakers Bureau Committee of the Auxiliary set up as the Speakers Bureau of the Society. All women's clubs are being contacted and asked to give some time on their programs to our present problems.

Mrs. F. H. Davis was elected president of the Woman's Auxiliary to the Lafayette Parish Medical Society when it was reorganized in 1948. Its major project in 1948 was a five hundred dollar contribution to the Cancer Society with funds raised by a style show sponsored by the Auxiliary. At a recent meeting Mrs. George M. Armstrong, Jr., parish visiting teacher, presented an informative talk on health needs of school age children. In May of each year the Auxiliary has a tea to which all of the senior girls of the parish are invited. Nurses and medical technicians address this group on the value of becoming nurses or technicians.

Auxiliary members interested in reading the testimony of Dr. Marjorie Shearon should write for Vol. V of the Hearings on S. 545 and S. 1320 of the 80th Congress to Mr. Ray Rodgers, Clerk, Senate Committee of Labor and Public Welfare, Washington, D. C. It is free.

# BOOK REVIEWS

*Manual of Leprosy*: By Ernest Muir, C. M. G., C. I. E., M. D., F. R. C. S., Edin., Medical Adviser, British Empire Leprosy Relief Association; Secretary, International Leprosy Association; Late Research Worker in Leprosy, School of Tropical Medicine, Calcutta. Baltimore, The Williams & Wilkins Company, 1948. Pp. 208. Price, $5.00.

The appearance of two works on leprosy, (the other by Dr. R. S. Cochrane), in about a year by English writers is probably due to reports of more promising drugs for treatment.

Dr. Muir has had broad experience with leprosy in many parts of the world and writes authoritatively on the subject. The present volume puts into a very small space the essentials of handling the disease, and is made up of twenty-seven chapters under three broad headings.

The first section, consisting of eleven chapters, is devoted to discussion of general features, including history and classification. The physician who has not kept in touch with developments in this field will find especially helpful the discussion of the tuberculoid type, which is illustrated by a number of useful colored pictures. The relation of the "uncharacteristic" form to the two main types (lepromatous and tuberculoid) is shown by a readily understood diagram.

The second section, consisting of nine chapters, deals with diagnosis and treatment. Dr. Muir has more confidence in the results of histologic examination than seems warranted by the frequently encountered unsatisfactory or contradictory reports on this phase of diagnosis. One can heartily agree with the author's low opinion of skin tests with lepromin, as an aid in diagnosis but he finds them of value in prognosis and type diagnosis. Chaulmoogra oil is preferred by Dr. Muir in the treatment of tuberculoid lesions, which of course are very likely to do well without any, or with no special treatment. He favors intradermal administration of the oil. Dr. Muir is impressed by the value of the sulfones in lepromatous leprosy and has used diasone chiefly. This drug, if further experience confirms its usefulness, has the advantage of being given by mouth, a factor that makes it convenient for use by physicians in private practice, and should go far to simplify treatment.

The chapter on surgery deals with several common surgical complications but makes no mention of tracheotomy for laryngeal obstruction. In the chapter on prognosis, Dr. Muir recognizes the great tendency of leprosy to spontaneous improvement, even in severe cases.

The third section, consisting of seven chapters, entitled "The Anti-Leprosy Campaign", will be of most interest and importance to health officers. Dr. Muir views the subject in a broad way. Clearly, in his opinion, it is not simply a matter of making a diagnosis of leprosy and consigning the patient to an isolation center. The Norwegian plan, based largely on home isolation, is spoken of with approval. Compulsory isolation is regarded as necessary only in endemic areas if suitable accommodations can be provided for all cases. Dr. Muir regards the distribution of immune and endemic regions in the United States as due largely to differences in standards of living and sanitation in various areas. "Leprosy consciousness," by which the author means fear of the disease by the general public, is recognized as of great importance in aiding in the control. Two chapters are devoted to leprosy surveys. One type of these is designated P. T. S. (Propaganda—Treatment—Survey), in which therapy and education are combined with case finding—as in tuberculosis campaigns—a very logical approach. Out-patient clinics are regarded as most helpful in such a program.

A chapter is devoted to "Health Children of Leprous Patients," and the Brazilian "preventoria" are favorably regarded. The final chapter in the book is a consideration of Social and Welfare Service, with reference especially to education, occupation, recreation, and spiritual elements. Nor are the dependants of patients forgotten; the family must be provided for when the bread winner is no longer in a position to provide support. An appendix deals with the preparation of the esters of chaulmoogra oil,—which seems superfluous—and another with "Intensive Surveys," which might be helpful.

One is pleased to note that Dr. Muir has avoided the use of the word leper, an omission that will be appreciated by patients suffering with leprosy.

Finally there is a bibliography arranged under the headings of the chapters of the text.

G. W. McCoy, M. D.

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*You and Your Doctor*: By Benjamin F. Miller, M. D., New York, Whittlesey House, 1948. Pp. 183. Price, $2.75.

This book attempts to bring to the lay citizen a frank discussion of group medical practice and other modern trends in American medicine.

For the "pilot physican," a well compounded mixture of the family doctor and the specialists, a three year postgraduate period is not excessive, particularly if through government participation, adequate salaries were provided for interns and residents.

Doctor Miller believes that group clinics for all areas would provide better diagnosis and treatment. These may be provided by guaranteeing the income of a local medical group on an actuarial basis, by an insured prepayment plan, or by a tax-supported plan, or any other workable variant.

Giving further responsibility to the government, he states that nursery schools should be provided for all families, regardless of financial status.

Federal support of medical science and practice, eventually extending to medical education, rounds out the author's all-inclusive program.

Though we may not be in accord with Doctor Miller's hopes and dreams, it is important that we know both sides of the issue.

EUGENE H. COUNTISS, M. D.

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*Psychobiology and Psychiatry*: A Textbook of Normal and Abnormal Human Behavior; by Wendell Muncie, M. D. 2nd Ed., St. Louis, C. V. Mosby Co., 1948. Pp. 620. Price, $9.00.

This is the second edition of a book which was first published in 1939. Because of the many subsequent changes, sometimes of a revolutionary nature, in our thinking about psychiatry, extensive editing and revision have been necessary. There were formerly four major divisions of the text; the fourth, which contained historical appendices, has been omitted, because this material is now generally available in most medical libraries.

Primarily written for the student, this is a textbook which "attempts to give a fair account of the conceptions, teaching, and working methods" of the Henry Phipps Psychiatric Clinic at the Johns Hopkins Hospital. Accordingly, the nomenclature of Adolf Meyer is used, but not insisted upon.

Perhaps the most valuable—and unique—single section is the first, entitled, "Psychobiography—The Study of Normal Behavior." This is devoted largely to the student's personality study, which is an excellent preparation in the technics of observation and understanding of a human being, in this case the student himself. The second section, "Abnormal Behavior—Pathology and Psychiatry," is liberally studded with case histories illustrative of the various pathologic processes described. The final section on "Treatment" furnishes a full introduction to the general bases of therapy and to the important therapeutic aids — including prefrontal lobotomy—available to the psychiatrist. The remainder of this section is then devoted to the more specific treatment of the various reaction states. The theories of psychoanalysis are perhaps unduly slighted, but with this possible exception, the book offers both student and general practitioner a reasonably well balanced survey of the enormous field of psychiatry and its related problems.

THEODORE F. TREUTING, M. D.

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*Essentials of Pathology*: By Lawrence W. Smith, M. D., F. C. A. P., and Edwin S. Gault, M. D., F. C. A . P., 3d ed., Philadelphia, The Blakiston Co., 1948. Pp. 764. Price, $12.00.

The authors are experienced pathologists and teachers. They have compiled a splendid collection of cases, which serve as illustrations for the pathologic processes discussed in their text. In most instances there are good photographs, or diagrams of the lesions. The material is presented in orderly fashion and the style is clear, concise and to the point. The microscopic descriptions of the lesions should be particularly helpful for medical students. Although "Essentials of Pathology" is not intended to be a reference volume in pathologic anatomy, it has a select bibliography at the end. The book is recommended for teaching of sophomore medical pathology when instruction is primarily of the single textbook type. If the teaching program is such that the students do not have to follow a particular textbook, but must acquire their information from various sources, we feel that some of the other books on this subject would be more advantageous.

G. M. CARRERA, M. D.

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*Diagnosis in Gynaecology*: By James V. Ricci, A. B., M. D., Philadelphia, The Blakiston Company, 1948, pp. 259. Price, $4.50.

The aim of this text is to teach the art of gynecological history taking and conducting a careful examination in order to arrive at a correct diagnosis. The author has attempted to stress analyzing, evaluating, and synthesizing signs, symptoms, findings and laboratory data so that the student may learn to think in terms of sound differential clinical logic.

Since this book is only intended to supplement the standard texts on gynecology, the chapters on anatomy, histology, and embryology might have been deleted as unessential.

The genital structures, and the various diseases which affect them, form the basis of presentation and chapter division. The medical student will find this practical approach most helpful.

EUGENE H. COUNTISS, M. D.

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## PUBLICATIONS RECEIVED

Bruce Publishing Company, St. Paul: Psychodynamics and the Allergic Patient, by Harold A. Abramson, M. D., F. A. C. A.

The C. V. Mosby Company, St. Louis: Clinical Case-Taking (Fourth Edition), by George R. Herrmann, M. D., Ph. D. Campbell's Operative Orthopedics (Vol. 1 and II) (Second Edition), J. S. Speed, M. D., Editor, Hugh Smith, M. D., Assoc. Editor.

Henry Schuman, New York: Doctors in Infamy, by Alexander Mitscherlich, M. D. and Fred Mielke.

Charles C. Thomas, Springfield, Ill.: Child Psychiatry, by Leo Kanner, M. D. Manual of Clinical Laboratory Methods (Fourth Edition), by Opal E. Hepler, Ph. D., M. D.

The Williams & Wilkins Company, Baltimore: Symptoms in Diagnosis, by Jonathan Campbell Meakins, C. B. E., M. D., D. Sc., LL. D.